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ON THE NATURE OF PINK DISEASE.

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IT has been said that Australian physicians consider themselves to have a special claim to pink disease (Rocaz, 1933). Whether this contention is justifiable remains for the medical historians to decide; but one fact is clear—the careful clinical study of the disease began in Australia, and such work remains as a significant contribution (Swift, 1914). Ten years ago it was suggested that "stress" was an ætiological factor in pink disease—that the condition resembled an alarm reaction of Selye (1946), with adrenal medullary preponderance and possible functional adrenal cortical insufficiency (Cheek, 1950). One was impressed with the craving for sodium chloride exhibited by some of these patients, and by their failure to withstand intercurrent infection, atmospheric heat stress or added electrolyte loss. Clinically, hypotonic solutions given intravenously were found to be deleterious. Hæmoconcentration was detected at the outset of the disease before anæmia became significant, together with a trend towards a low plasma sodium concentration (Cheek and Stace, 1951; Cheek and Hicks, 1951; Stoneman, 1958). Efforts to expand extracellular volume by the use of desoxycorticosterone and sodium chloride were rewarded on occasions by dramatic clinical improvement. These early observations are reviewed elsewhere (Cheek, 1953).

At the time when this work was in progress, Warkany and Hubbard of Cincinnati (1948), and Fanconi and Botsztejn of Zurich (1948) found mercury in the urine of patients with the disease. Fanconi considered the disease a neuro-allergy to mercury, while Warkany regarded the condition as an "idiosyncrasy" to mercury "just as some persons react with asthma or hay fever to environmental conditions well tolerated by others". The involvement of mercury as an ætiological factor was not entirely new. Zahorsky of St. Louis (1922) stated that he was inclined to the view that calomel was the causative agent. Because he could not obtain a history of calomel ingestion in every patient with the disease, he gave the matter no further consideration. In 1935, Lesesne Smith of South

Carolina was strongly impressed with a relationship between calomel and pink disease, and influenced Davison to suggest mercury as the ætiological factor in the 1938 edition of his current text, "The Complete Pediatrician" (1938). If a normal infant is given calomel, the excretion of mercury in the urine is capricious (Zellweger Wehrli, 1951), being present one day and not the next. Furthermore, mercury is selectively taken up by renal tissue (Greif *et alii*, 1956), just as lead sequestrates in bones. For these reasons it has not been possible for every worker to detect mercury in the urine of all children with pink disease.

It is clear, however, that mercury is an ætiological factor; but it has been far from clear how mercury acts to produce the clinical picture of pink disease. It is the purpose of this brief review to demonstrate the probable role of mercury and to draw attention to a possible second ætiological factor — stress (or sympathetic activation).

From the time of the earliest writings concerning pink disease, a disturbance of autonomic function has been anticipated (Feer, 1923). The hypertension, tachycardia, sweating and hypotonia all supported such a view. Day *et alii* (1939) published experimental evidence that sympathetic activity was maximal in the disease; they showed that the sympathetic division of the nervous system was incapable of further stimulation. Vulliamy (1952), by studying skin temperatures and the action of ganglion-blocking agents, produced important evidence that widespread vasospasm existed, owing to increased sympathetic tone. Bower (1954) confirmed this work, and also confirmed the report by Gillespie (1952) of the achieving of dramatic clinical results by the use of antisympathetic drugs. Peterson and Laughmiller (1954) made similar observations concerning antisympathetic drugs. Other investigations (Cheek, 1957) revealed an increase in venous pressure, reduction of blood volume and, in one case carefully studied, an increase in basal metabolic rate. Contraction of blood volume and increase in venous pressure are also directly in line with sympathetic stimulation (Eckstein and Hamilton, 1957; Freis and Rose, 1957; Finnerty *et alii*, 1958). A preponderance of sympathetic activity would seem to be an established fact in pink disease.

Earlier work (Cheek, 1951) revealed no gross rise in blood catecholamine level in pink disease; but the method of assay was primitive in comparison with recent methods for the precise determination of blood epinephrine and nor-epinephrine contents (Cohen and Goldenberg, 1957; Jones and Blake, 1958). Of more importance was the work of Farquhar *et alii* (1956), who showed that the "sympathin" excretion in the urine was not elevated. *A priori*, it seemed that the predominance of the sympathetic activity might rest in the tissue response to epinephrine (as it does in thyrotoxicosis—Brewster *et alii*, 1956) or in an interruption in the destruction of the hormone.

Young male rats, five to six weeks old, were given 10 mg. of calomel (mercurous chloride—the preparation used in teething powders) by stomach tube, and one injection (0·5 mg.) of epinephrine in oil. Control animals were given either calomel *per se* or epinephrine *per se*, or no medication (Cheek *et alii*, 1959). Some 12 hours later it was noticed that rats given calomel *plus* epinephrine had sustained blueness in the extremities and a definite coldness to the feel. The insensible water loss was greater than that of the control rats, and the hæmatocrit was significantly higher. Likewise, there was a 15% loss of body chloride. Hypertension was greatest in the rats receiving calomel and epinephrine, and persisted for a longer time. Perhaps the most interesting finding was the demonstration that the young rat given calomel responded to a small intravenous dose of epinephrine with tachycardia, while a control rat developed bradycardia or no change in pulse rate.

These experiments strongly suggested that calomel could potentiate the tissue response to epinephrine. At the time of presentation of these data (Cheek, 1958), it was not known how mercury acted to alter epinephrine metabolism. In addition, an indirect effect was also worthy of consideration, since histological examination of the kidney revealed a lesion in the rats that received calomel plus epinephrine which was not seen in other control rats. The lesion was an area of degeneration situated mainly in the outer area of the renal medulla (the upper region of Henle's loop)—an area suspected by some pathologists as a critical site with respect to hypertension (Muirhead,[1] personal communication).

Recently, Axelrod and Tomchick (1958) demonstrated that the enzyme concerned with the inactivation of epinephrine in the body is methyl transferase (catechol O-methyl transferase). For this enzyme to work, a substrate, S adenosyl methionine, is necessary, and this substrate contains sulphydryl groups. Mercury (or calomel) will inhibit or remove sulphydryl groups, so that methyl transferase can no longer function. Here, then, is one means by which calomel can potentiate sympathetic activity.

In further experiments (Cheek and Wu, in the press), using modern methods of epinephrine determination, it was found that small amounts of calomel would raise the concentration of epinephrine in the blood of rats, and that exposure to cold stress would increase the level 400% above that of control rats.

If calomel acts in a similar manner in the infant, it is clear that activation of the whole sympathetic system can occur, and that the presence of coincidental "stress", whether environmental or due to previous illness, will enhance the whole process. Thus attention is directed back once more to the adrenal gland. As was emphasized by Ramey and Goldstein in their recent review of adrenal function (1957), if sympathetic activity overrides adrenal cortical activity, there is failure of adaptation or failure to withstand added stress. Since sympathetic activity is relatively greater in the infant, it is not surprising that pink disease is limited to the pædiatric age groups.

It would seem clear now why the 17-ketosteroid excretion is raised in this disease (Cheek, Hetzel and Hine, 1951). Since more water can be lost in sweat than in urine in this disease, the measurement of urine 11-oxysteroid excretion is of little value as an assessment of the total excretion of 11-oxysteroid. The changes in fluid and electrolyte balance in pink disease lend further credence to the view that functional adrenal cortical insufficiency exists.

In pink disease there is a loss of body chloride and extracellular volume (Cheek, 1957), even up to 30%. There is also an increase in red-cell water content, which probably represents a general trend towards increase in cell hydration in all tissues.

Calomel when given to rats causes loss of body chloride and sodium contents and gain in cell potassium content (Cheek, 1957; Cheek *et alii*, 1959). Calomel is a mercurial diuretic of bygone days. The increase in body potassium is of interest, since Gamble jr. (1959) has recently shown that mercury causes excessive binding of potassium by the mitochondria of the cell. Mercurhydrin, the diuretic, when given to rats, causes loss of body chloride and sodium and an increase in cell hydration. Mercurial diuretics supersede the sodium-retaining action of steroids at the renal level (Weston *et alii*, 1952). In a patient with pink disease it was found that desoxycorticosterone did not cause sodium retention (Cheek, 1954). Furthermore, it is noteworthy that salt and steroid administration produced dramatic clinical results in long-standing cases (Cheek and Hicks, 1950; Murray, 1950; Forman, 1951)—results contrary to the natural course of the disease — while later work and more experience with patients, some whose illness was of shorter duration, demonstrated a failure to produce such dramatic results (Cheek, 1951; Williams *et alii*, 1951). The failure of salt and steroid to cause a weight increase in most patients now finds explanation. Furthermore, the study of weight change during the course of the disease (Colver, 1955) is compatible with a rela-

[1] Professor E. E. Muirhead, University of Texas Southwestern Medical School, Dallas, Texas.

tively sudden fluid and electrolyte loss, which is followed by a plateau of sustained low weight; then, after several weeks, a gradual gain of weight occurs with recovery. Thus the changes in weight and in fluid and electrolyte balance find explanation on the grounds that mercury or calomel is acting at the renal level. Since extracellular volume is not maintained, the concept of functional adrenal cortical insufficiency would seem justifiable.

In summary, investigations over the last decade into the physiology of pink disease lead to a definite concept relating to the nature of this disease. If sympathetic activity is enhanced in the infant — for example, by previous illness or other environmental factors — this state will be perpetuated and augmented in the presence of calomel or other inorganic mercurials. Mercury can block the destruction of epinephrine. Furthermore, calomel can act as a mercurial diuretic and can reduce body sodium chloride content and extracellular volume, which in turn will activate the adrenal medulla and cortical system. This system may or may not act effectively at the renal level to conserve sodium chloride, since mercury blocks the action of steroids on the kidney. The sympathetic division of the autonomic nervous system is more active in the infant than in the adult, and one is not surprised that pink disease is limited to the early period of life.

Adrenal physiologists now recognize that if sympathetic activity overrides adrenal cortical activity, a failure of adaptation results. This disease is principally one of endocrine dysfunction, initiated by pharmacological actions of mercury. There would seem to be two ætiological factors involved — mercury (inorganic) and stress.

Removal of as much mercury as possible from the body would seem important (Elmore, 1948; Bivings and Lewis, 1948; Epstein, 1950), together with the exhibition of antisympathetic drugs and efforts to restore extracellular electrolyte. The use of steroids would seem to be justifiable, in so far as they may restore adrenal medullary-cortical balance and help to correct electrolyte imbalance. However, complete removal of mercury from renal tissue is probably difficult, and effective actions of steroids may be offset at the renal level.

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THE VIRUS ÆTIOLOGY OF EPIDEMIC EXANTHEM AND POLYARTHRITIS.

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AN epidemic of exanthem and polyarthritis occurred in Mildura and neighbouring towns in the Murray Valley during April and May of 1956. Investigations at that time by Anderson and French (1957), by Fuller and Warner (1957), and by Wilson (1957), revealed the epidemic to be extensive, involving between one and two thousand cases in Mildura alone. The nature of the epidemic suggested that it was probably due to an arthropod-borne virus. Anderson and French studied 36 cases intensively in an attempt to discover the ætiology. No agent was isolated. Not until recently has serological evidence accumulated to point more directly to a group A arthropod-borne virus as the cause.

The clinical disease was mild, and was characterized by a rash which in many cases covered the entire skin surface, beginning as discrete macules which became papular and, in a few instances, progressed to small vesicles. Some patients had petechiæ and two an enanthem. Twenty-three patients had joint pains, primarily in the small joints of the extremities; these usually preceded the rash, in one case by as much as ten days. Joint swelling was rare, and occasionally effusion occurred. Joint symptoms lasted from two to 28 days, and in one case persisted for eight months. Few patients complained of fever, and the highest recorded temperature of those examined was 100·8° F. Five patients developed tender and painful glands. Strikingly, six of the 36 had tenderness of the palms and soles, and five had paræsthesiæ. Leucocyte counts varied between 2800 and 7000 per c.mm. during the acute phase of illness. During the epidemic, many patients complained of typical joint or muscle pains without a rash. It was thought that these were due to the same disease.

The disease was confined to the valleys of the Murray River and several of its tributaries, which near Mildura flow through semi-arid country. Anderson and French considered the incubation period to be the interval between infection and first appearance of the rash. On the basis of the length of time that elapsed between patients leaving the epidemic area and becoming ill, these authors estimated the incubation period as approximately ten to eleven days. It was considered significant that 27 patients, all of whom were closely questioned, had visited the river banks one to two weeks before onset.

Other similar epidemics in Australia and in the islands of the South-West Pacific have been reported since 1928, when Nimmo investigated an epidemic of rash and arthritis among country dwellers of New South Wales. Other outbreaks were reported by Halliday and Horan in 1943, and by Harris in 1943-1944 among soldiers in the Northern Territory; by Sibree in Queensland and by Goswell in the Northern Territory in 1944; by Weber *et alii* and McCarry *et alii* among U.S. troops in the South Pacific in 1945; and by Dowling in 1945 in Queensland. The outbreaks in Queensland occurred among troops training in the jungle on the Atherton Tableland, inland from Cairns (Sibree and Dowling, personal communications).

The causative agent or agents have not been isolated, although at least three groups of investigators (Anderson and French, 1957; Fuller and Warner, 1957; Halliday and Horan, 1943) made exhaustive efforts in this regard. These epidemics resembled an outbreak of acute virus polyarthritis which occurred in Tanganyika during 1952 (Robinson, 1955). An arthropod-borne virus, subsequently named Chikungunya, was isolated from man and mosquitoes and identified by Ross (1956) as the probable cause of the polyarthritis. Recent studies by Spence and Thomas (1959) have shown that this agent belongs to Casals' group A of arthropod-borne viruses. In marked contrast to the Tanganyika outbreak, in which there was sudden onset of severe arthritis and fever followed by a rash, the Australian disease was mild, with gradual onset. Convalescent sera obtained from the patients in Mildura during the 1956 epidemic were devoid of Chikungunya antibody. However, four other group A viruses were inhibited by convalescent, but not by acute, sera in hæmagglutination-inhibition (HI) testing. Thus, it

TABLE I.

Epidemic Polyarthritis and Rash: Results of Serological Testing of Patients who Developed Detectable Antibodies in Convalescence.

Patient.	Antibody Titres.										
	Hæmagglutination-Inhibition Test.					Complement Fixation Test.		Neutralization Test[2]			
								AMM 2021 Virus.		AMM 2354 Virus.	
	Semliki Forest Virus	AMM 2021 Virus.	AMM 2354 Virus.	Mayaro Virus.	Chikungunya Virus.	AMM 2021 Virus.	Nine Other A Group Viruses.	MR.	AST.	MR.	AST.
Bay.:											
Acute stage	0[1]	0	—	0	0	0	0	8/8	2·3	—	—
Convalescent stage ..	80	10	—	10	0	4	0	8/8	2·5	—	—
McD.:											
Acute stage	0	0	0	0	0	—	—	8/8	4·1	7/8	4·0
Convalescent stage ..	20	20	20	0	0	—	—	2/7	7·0	0/8	9·0
Flem.:											
Acute stage	0	0	0	0	0	—	—	8/8	2·3	8/8	3·4
Convalescent stage ..	20	0	40	10	0	—	—	6/7	3·6	8/8	3·9
Win.:											
Acute stage	0	0	0	0	0	—	—	8/8	2·0	8/8	3·4
Convalescent stage ..	20	0	80	80	0	—	—	7/7	3·0	2/8	8·1
Kle.:											
Acute stage	0	0	0	0	0	—	—	8/8	2·3	6/8	5·3
Convalescent stage ..	80	10	320	40	0	—	—	7/7	2·3	3/8	7·8
Geb.:											
Acute stage	0	0	20	0	0	0	0	7/8	4·3	6/8	5·0
Convalescent stage ..	80	20	160	40	0	16	0	0/7	9·0	0/8	9·0

[1] Negative at 1 : 10 dilution; numbers indicate reciprocal of highest serum dilution reacting with antigen.
[2] MR, mortality ratio: number dead/number inoculated; AST, average survival time (days).

TABLE II.

Survey for Group "A" Antibodies among Normal Persons Residing in Australia and New Guinea.

Collection Site.	Age. (Years.)	Sex.	Antibody Titres.					
			Hæmagglutination-Inhibition Test.				Neutralization Test.[1]	
			AMM 2021 Virus.	AMM 2354 Virus.	Mayaro Virus.	Semliki Forest Virus.	AMM 2021 MR.	AST.
Brisbane	—	—	10	0	—	20	5/7	4·7
	—	—	20	10	—	80	0/7	9·0
	—	—	0	0	—	10	0/6	9·0
	—	—	0	0	—	0	0/7	9·0
Cairns	—	—	40	10	—	80	0/7	9·0
	—	—	40	20	—	320	1/6	8·7
Orange, N.S.W.	—	—	0	10	—	0	7/7	3·0
	—	—	20	10	—	80	0/5	9·0
Popondetta	32	M.	20	20	0	—	—	—
	30	M.	0	0	0	—	—	—
	30	M.	40	20	0	—	—	—
	33	M.	0	0	0	—	—	—
	29	M.	0	20	0	—	—	—
	35	M.	20	20	0	—	—	—
	31	M.	0	0	0	—	—	—
	30	M.	40	20	0	—	—	—
	30	M.	0	0	0	—	—	—
	35	M.	40	0	0	—	—	—
Kokoda	33	F.	0	0	0	—	—	—
Tufi	30	F.	10	0	0	—	—	—
New Britain	—	—	10	0	0	—	—	—
Sepik Valley	5	M.	0	0	0	—	—	—
Aramia River	7	—	160	40	10	—	—	—
	9	M.	40	40	10	—	—	—

[1] See footnote to Table I.

became evident that the disease was not caused by Chikungunya, but by a virus related to it. This finding prompted a more detailed study of these sera.

Methods and Materials.

Sixteen pairs of acute and convalescent human sera as well as sera from seven water-rats and 18 chickens, were collected in Mildura during 1956. In addition, 50 samples of human serum from a survey in Australia and New Guinea were available for testing.

Serum samples of 1 ml. volume each were stored at −20° C. for two years, and then lyophilized and rehydrated prior to use.

HI testing by the methods of Clarke and Casals was performed in lucite plates, with the use of suckling mouse brain antigen extracted by the sucrose-acetone method. Antigens treated with protamine sulphate were used in some cases.

Complement-fixation (CF) testing was done in plastic plates by microtechnique, by means of a modification of the method described by Fulton and Dumbell (1949). Mouse-brain antigen extracted by the sucrose-acetone method and "Veronal" (pH 7·3) diluent were used. The sera were inactivated for 20 minutes at 60° C.

For neutralization testing, sera from infected suckling mice were used as a virus source. The virus preparations were diluted in 0·75% bovine plasma albumin solution, at pH 7·2, to contain approximately 100 mouse LD_{50}. Swiss mice, aged one to two days, were given virus-serum mixture by intraperitoneal injection, 0·03 ml. per animal. These mice were observed for nine days.

Experimental Data.

The viruses used in testing were Chikungunya, Semliki Forest, Mayaro, Middelburg, Venezuelan Equine Encephalitis, Western Equine Encephalitis, Eastern Equine Encephalitis, Sindbis, AMM2021, and AMM2354. These last two viruses were isolated from culicine mosquitoes in Malaya by the members of the U.S. Army Medical Research Unit, and supplied for testing by Major Edward Buescher of the Walter Reed Army Institute of Research. As is shown in Table I, in six of 16 serum pairs there was a rise in antibody titre to one or more of these viruses. These results indicate clearly that infection with a group A arthropod-borne virus was probably associated with some of the cases of polyarthritis. From the pattern of inhibition, however, it is impossible to make a more specific diagnosis. In general, infections with a group A virus

TABLE III.

Epidemiological and Clinical Observations of Nine Epidemics of Rash and Polyarthritis.

Author.	Date.	Area.	Number of Cases.	Clinical Manifestations.			White Cell Count.	Culicine Mosquitoes Noted.	Suspected Incubation Period.
				Rash.	Arthritis.	Lymph-adenopathy.			
1. Nimmo (1928).	March and April, 1928.	New South Wales.	100	+	+	Not stated.	Not stated.	+	Not stated.
2. Halliday and Horan (1943).	October, 1942, to January, 1943.	Northern Territory.	105	+	+	+	Normal to increased.	+	Not stated.
3. Harris (1944).	November, 1943, to April, 1944.	Northern Territory.	65	+	+	+	Normal.	Not stated.	Less than 10 days.
4. Goswell (1946).	Early 1944.	Northern Territory.	51	+	+	+	Normal.	+	Not stated.
5. Sibree (1944).	February to March, 1944.	Queensland.	28	+	+	+	Normal.	+	Not stated.
6. Weber *et alii* (1946).	December, 1944, to July, 1945.	Schouten Island, New Guinea.	48	+	+	+	Leucopenia.	Not stated.	Not stated.
7. McCarry *et alii* (1946).	Not stated.	Bougainville.	Not stated.	+	+	+	Not stated.	Not stated.	Not stated.
8. Dowling (1946).	February to April, 1945.	Queensland.	94	+	+	+	Normal.	+	Not stated.
9. Anderson and French (1957), Fuller and Warner (1957) and Wilson (1957).	April to May, 1956.	Murray Valley.	1000+	+	+	+	Leucopenia.	+	10–11 days.

lead to the development of HI antibodies, not only to the infecting agent, but also, though to a lesser extent, to closely related agents. Each group A infection produces, as a rule, a very distinctive pattern, from which a specific diagnosis can usually be made. The results shown in Table I indicate no distinctive pattern. There was a rise, not only in HI antibodies, but also in CF and neutralizing antibodies. In the only two pairs of sera tested by CF, both acute phase sera gave negative results, whereas the convalescent sera fixed complement in the presence of an antigen prepared from AMM2021, but not with antigens prepared from the nine other members of group A.

In the neutralization tests, which were performed with the two Malayan agents (AMM2021 and AMM2354), all the acute phase sera gave negative results, whereas four of the six convalescent sera showed definite evidence of protective antibodies to one or the other or to both the viruses.

From these results, it is clear that by all three tests evidence was obtained for a rise in antibodies to members of group A arthropod-borne viruses.

Twenty-four human sera, from normal individuals in New Guinea and neighbouring islands, as well as from the north-east coast of Australia, were surveyed for the presence of group A antibody. The results are presented in Table II. Seventeen of the 24 sera from New Guinea and Australia had evidence of group A antibody by HI testing, and six of the eight Australian sera showed neutralizing antibodies in a neutralization test with AMM2021 virus. In addition, 26 sera from Bougainville Island were tested, but showed little evidence of group A virus activity. The sera from New Guinea and neighbouring islands were collected during 1956-1957, and those from Brisbane, Cairns and Orange in 1951-1952.

HI testing of the sera from seven water-rats and 18 chickens from the vicinity of the Murray River, which were bled shortly after the epidemic in 1956, gave negative results.

Discussion.

The fact that six of 16 patients developed antibodies to group A arthropod-borne viruses during attacks of polyarthritis and rash in Mildura in 1956 is presumptive evidence that the epidemic was caused by an arthropod-borne virus related to those used in serological testing. The pattern of HI response, however, is not one that would be expected from any given single virus among those used in testing. Accordingly, it seems probable that the virus which caused the outbreak of rash and polyarthritis, if isolated, will be found to differ from the existing identified members of group A. Possibly more than one group A virus is active in Australia.

Table III summarizes the clinical and epidemiological observations during the nine outbreaks of polyarthritis and rash in Australia and its vicinity. All have occurred during the wet months of the year; all have been clinically mild. The predominant arthropod seems to have been the culicine mosquito, which was specifically noted as prevalent by six of the investigators, although the March fly has also been suspected in some epidemics.

Most of the epidemics reported have occurred in the northern areas of Australia or in the islands to the north, especially among troops who were, presumably, non-immune, and had been newly introduced to the area. Anderson and French (1957) postulated that the virus might be endemic in northern Australia, and had occasionally extended southwards when the arthropod vector was abundant and other conditions were favourable for spread—a situation analogous to that with Murray Valley encephalitis as reported by Anderson in 1954. The fact that a large percentage of the people surveyed in New Guinea and northern Australia had group A antibodies lends credence to this hypothesis.

Summary.

Patients suffering from arthritis and rash in Mildura in 1956 developed antibodies to members of Casals' group A arthropod-borne viruses. The causative virus has not yet been isolated in Australia, and may be a new virus. Sera from normal persons in New Guinea and in north-eastern Australia contain HI antibodies to group A viruses.

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THE HEALTH OF IMMIGRANTS: SOME OBSERVATIONS FROM GENERAL PRACTICE.

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IN this general practice, in a closely settled industrial area of Adelaide, about one patient in four is an immigrant from the continent of Europe; there has therefore been a good opportunity to observe some of the hazards to health that may be related to immigration.

Immigrants may be classified in various ways: into racial, ethnic or national groups; into linguistic, religious or economic groups. They may be divided into those who left their homeland of their own free will, and those who left under duress: whether they were displaced or stateless persons, refugees from persecution or members of minority groups seeking a greater degree of freedom than

could be found in their country of origin, or whether they sought better economic conditions or better opportunities for their children.

Some have come here alone, some with wives and families. Some have brought wives and families or betrothed ones to join them after they had established themselves in this country; others have married people already resident here. Some have retained many of the tangible and intangible characteristics that symbolize their homeland; others have shed these things and become assimilated into their new environment to a degree which makes it difficult to distinguish them from native-born Australians.

Though little published work on the health of immigrants has appeared in Australia, there is an extensive body of literature on the sociological and demographic aspects of migration, and on the assimilation of immigrants (Borrie and Packer, 1953, 1958). Borrie (1954) gives this definition of assimilation:

> Assimilation is a psychological, socio-economic and cultural process, resulting in the progressive attenuation of differences between the behaviour of immigrants and nationals within the social life of a given country.

Can a person be uprooted from the homeland and set down in a new country—new climate, new social customs and economic conditions, to say nothing of a new language—without some deleterious physical or psychological consequence? The psychological change implied in the definition of assimilation may be drawn out over a period of years; or assimilation may fail, with other, perhaps worse consequences, especially when these are considered as a problem of mental health. During the process of assimilation, immigrants may suffer the same diseases as their fellow men. Are they also particularly prone to some diseases because they are immigrants? Can any forms of sickness be correlated with a socio-economic classification of immigrants, along the lines suggested above? Very large numbers of people would have to be studied before these questions could be answered. It is not possible to base generalizations on the limited number of observations that can be made in one general practice. For this reason no classification of health and sickness in relation to immigrant status will be attempted here. The generalizations that are submitted can be put only in the form of clinical impressions, and are submitted only in the hope that they will stimulate further inquiry, especially in general practice. Cases falling into certain broad groups will be described. Those which illustrate the possible consequences of a relatively low standard of hygiene among certain classes of immigrants have not been included. It is hoped to make this the subject of a separate report, which, to have any meaning, would have to include evidence gathered from other sources than one general practice.

An illness clearly related to immigrant status is one not naturally occurring here, such as Mediterranean anæmia, which is occasionally seen in southern European patients attending the ante-natal clinics of maternity hospitals throughout Australia. This disease is less often seen in general practice, partly because of the difficulty in establishing the diagnosis in latent cases. One suspected case has been seen in this practice, but the patient defaulted before detailed investigations could be made.

Some immigrants have brought with them on their journey to Australia an intestinal parasite, which could become disseminated here if standards of sanitation were to be neglected.

CASE I.—A male patient, aged 25 years, from East Germany, arrived in Australia in 1955. He was seen in July, 1957, when he complained that he had recently seen segments of tapeworm in his motions. He had lost one and a half stone in weight in about two months, had lost his appetite, and felt weak and ill. He had been treated on two previous occasions for tapeworm, the last time about a year before he left Germany. Apparently extract of male fern was used on both occasions. Examination showed him to be extremely thin, to the point of emaciation; and segments of *Tænia saginata* were seen in the stools. He was treated with "Atebrin" tablets and magnesium sulphate. The result was a tumultuous evacuation of the bowels with many feet of worm. Unfortunately the head was not found, but may have been passed in a public lavatory which he was obliged to visit on his way to the laboratory of the Institute of Medical and Veterinary Science in Adelaide. He has remained very well since then and has put on over two stone in weight, so the presumptive evidence that he passed the head of the worm is reasonably strong.

The impact of a new language and new customs or a new set of traffic rules and regulations can have unfortunate consequences. Traffic-conditioned reflexes acquired in one country may operate adversely in another, as many of us have discovered on first visiting the continent of Europe or North America.

CASE II.—A male patient, aged 25 years, from Hamburg, Germany, had been in Australia for three years when in February, 1959, he was riding his motor-cycle along a main road and a car came out of a side street on his right and swung in, in front of him. He suffered a momentary indecision as to whether he should veer to the right or the left of it, with the result that he collided with the rear of it, sustaining multiple lacerations and abrasions.

Industrial accidents, such as injuries due to objects dropped onto the feet, attributable to misunderstandings between workmen obliged to converse by signs or half-understood phrases, are so common that one or more is likely to be seen in a morning spent in any busy casualty surgery.

There is a story, probably apocryphal, about the Italian who attended with two black eyes and the story that he had merely called *"abbastanza"* (enough) to his Australian workmate, who was helping him load a truck. Apparently he had been misinterpreted.

There are many other ways in which unfamiliarity with local customs can be dangerous. An unforgettable emergency that occurred eight years or so ago in a small country town may be related here.

One sunny afternoon in early May, a newly arrived family from Latvia went mushrooming. At home at the end of the afternoon they carefully divided the mushrooms from the puffballs, then threw away the mushrooms and cooked and ate the puffballs. When seen an hour or so later at the local hospital, mother, father and two small children were all very sick and sorry for themselves. They had some of the features of muscarine overdosage, but luckily for them vomiting was by far the most prominent symptom, and the worst of the poison was rapidly eliminated. It may be assumed that they did not make the same mistake again.

Three cases of active tuberculosis in immigrants have been seen in this practice in the last two years. All three patients were young, industrious people whose lost working time has been a preventible calamity in their lives.

CASE III.—A male patient, aged 29 years, from Benevento, Italy, arrived in Australia in 1953. He had been a farm worker in Italy, but he settled in an industrial suburb of Adelaide and worked as a press operator in a factory. He worked extremely hard, with no holiday from the time of his arrival. He did a lot of overtime, and took on odd jobs, mostly gardening and the like, over week-ends when no overtime work was available. His objective was the usual one of providing a home for his wife and two small children as quickly as possible. In June, 1957, he complained of chest pain, shivers and sweats, and a slight cough. Clinical and radiological examination revealed a large pleural effusion. A Mantoux test (1:1000) gave a strongly positive result. He was admitted to the Royal Adelaide Hospital, where, after further investigation, the diagnosis of primary tuberculous pleural effusion was made. He was treated with streptomycin and isoniazid, and eventually returned to work about a year after his original illness.

CASE IV.—A female patient, aged 25 years, from the Austrian Tyrol, arrived in Australia in 1953. She had lived in a small village near Innsbruck and had never had a day's illness in her life before she left to come to Australia. From the time of her arrival she worked without a break in a small factory making leather goods. Her husband was a shift-worker, mostly on night-shift. Both worked overtime and did odd jobs, she, for example, taking evening work as a waitress in one of the "continental" restaurants in Adelaide. They had paid for a block of land and lived in an asbestos hut, containing good quality furniture they had bought. They had no children, but she had had a miscarriage, perhaps self-induced, about two years after their

arrival in Australia. She was seen in April, 1958, when she complained of a cough with mucoid sputum present for several months. The result of clinical examination was unremarkable, but X-ray films showed tuberculous infiltration in the right hilar region and her sputum contained tubercle bacilli. After ten months of medical treatment her disease was rendered quiescent.

CASE V.—The third immigrant patient with tuberculosis seen in this practice was a young man who had several hæmoptyses while studying for an examination. He ignored these until his examinations were over, when he presented for investigation. X-ray examination of his chest showed that he had bilateral infiltration and a small cavity, and his sputum contained tubercle bacilli. He had had a pleural effusion in Holland some years before coming to Australia. His father (Case IX) is also under observation, having had active tuberculosis while a prisoner of war in Dachau concentration camp. His disease has remained quiescent.

Any discussion of the subject of tuberculous infection in immigrants can become charged with emotional overtones. However, a controlled investigation, carried out in Britain by Hess and Macdonald (1954), showed the incidence of tuberculosis to be very high among Irish immigrants, who had most commonly come from a rural background to live under conditions of extreme overcrowding in the industrial cities. Here they would appear to have fallen easy prey to the tubercle bacillus.

An editorial writer in the *British Medical Journal* (1958) has pointed out the need to distinguish between Mantoux-negative immigrants, such as the majority of the Irish entering Britain, and those others who carry the tubercle bacillus into the country with them. No satisfactory method has been found to protect and detect respectively these two classes of people. Here, where the requirements for entry into the country are stricter than in Britain, it should be possible to make greater use of tuberculin skin tests, followed, when indicated, by B.C.G. vaccine. The difficulties of detecting cases of active tuberculosis have been briefly discussed by Burgess (1952), who found that 10% of people presenting for examination at migrant selection centres in Europe had radiological evidence of tuberculosis, probably active.

It should be possible to eliminate or greatly reduce this hazard to health, for, in this district at any rate, immigrants have the same tendency as the Irish in Britain to live very often under congested conditions with poor standards of hygiene and sanitation. Many also work harder than is compatible with the maintenance of good health, and may thus be rendered susceptible to infection, or to reactivation of quiescent disease.

Some features in the history of the girl in Case IV have been seen often enough to lead to the clinical impression that this is not an uncommon pattern of behaviour among the hard-working, thrifty and in some ways far-sighted young migrants from countries like Germany and Holland. Many similar married couples have been encountered, all with the common objective of owning their own home as soon as possible, and sacrificing all else, including the conception and rearing of children, to this objective. Several have been asked whether they would have been obliged to postpone having children had they not emigrated, but no satisfactory answers have been received. Through choice or necessity, they are paying for their homes by private mortgage, rather than buying them on long-term loans which should allow them enough ready cash for what some of them, at any rate, regard as the luxury of children. It must be pointed out, however, that for every one in this category there are probably many who are more fortunate, or have planned better, and who, by buying their home through the State Housing Trust, or by means of some other long-term housing loan, have been able to contribute children as well as labour in return for their passage to Australia.

CASE VI.—A female patient, aged 25 years, from Berlin, had been in Australia since 1954, having married in Germany a week or two before emigrating. She was seen in January, 1959, when she complained of lassitude, loss of over a stone in weight in a month, complete loss of appetite and severe insomnia. Examination, including X-ray examination of the chest, showed only that she looked more like 35 years of age than 25. Since her arrival in this country she has worked at a factory where she assembles wire springs. For the last three years she has been working at home as well; she and her husband have been able to pay the deposit on a home, which they are paying off at the rate of £15 weekly on a privately arranged mortgage with the previous owner. Four rooms of this home are rented to single men, for whom the patient cooks and launders. The home is spotless and well furnished. She would like to have children, but her husband insists they must pay off what is owing on their home first. Her husband is a stern, autocratic person whose behaviour would probably have been essentially the same whether he had emigrated or not. She, on the other hand, is certain she would not have tolerated childlessness, nor would she have taken so kindly to the life of drudgery she leads, had she remained at home in Germany. At the time when they contracted to buy their home she, and probably her husband as well, knew nothing of the long-term loan arrangements that are available for the purchase of a home. It is idle to speculate whether Australia would have gained their offspring had they known of these arrangements.

Another "pattern" of behaviour in relation to childbearing is illustrated by the following case history, one of three very similar ones in this practice in the last two years.

CASE VII.—This woman was 34 years old in 1957 when she came out from Italy to join her fiancé, who had been here for some six or seven years. After about eighteen months of marriage, husband and wife came along to see if there was any reason for their failure to have a child. She did in fact conceive a child shortly after investigations had been begun, but their hopes were soon dashed, for after three months she miscarried. There has been no further pregnancy so far.

Two other cases of marriage delayed in this way through force of economic circumstance, with difficulty in conception shortly followed by miscarriage, are identical in all respects with this one. In each case the man had been living and working in Australia for a number of years, and had not been joined by his wife until he had a good home waiting for her—a most praiseworthy aim, which makes it particularly sad to observe the sequel. These marriages would not have been so long delayed had the men remained in their homelands.

Up till the end of 1956, no accurate survey of the incidence of neurosis and psychosis in immigrants had been possible in Australia (Redshaw, 1956). The assessment of mental health, and estimation of the incidence of neurosis and psychosis among immigrants, have proved impossible tasks under the uncontrolled conditions of general practice. The presence of a large population of transient residents in this district makes accurate mathematical calculation impossible. Furthermore, it is in these transient residents that language difficulties most often occur. The absence of close *rapport*, which occurs when interviews have to be conducted with an interpreter or in school-book German or Italian, makes assessment of the mental health of a patient very difficult indeed.

In 1958, approximately 500 "New Australians" were seen in this practice. Of these, some 50 were suffering from neurosis, or from "stress" diseases such as peptic ulcer and hypertension. There were three mildly psychotic patients, and one severely psychotic patient who required certification was seen.

These figures must not be taken as an indication of the prevalence of mental ill-health among immigrants, for the reasons given above. Other factors, such as chance, choice of doctor by patients, and the fact that many immigrants in closely settled areas such as this will by-pass the general practitioner by going to a public hospital, will invalidate any attempted calculation of disease incidence.

Working under better conditions, others have experienced similar difficulties. Weinberg (1955) was able only to say that adjustment of immigrants (in Israel) to their new living conditions was dependent upon their state of mental health, rather than upon whether their migration had been voluntary or involuntary. In order to obtain this result, an extremely complex series of investigations was

carried out upon a small and somewhat preselected series of immigrants. Nothing on this scale would be practicable in a normal population sample.

What effects may environmental factors have upon the incidence of neurosis or stress disease? No authoritative answer is possible from the limited experience obtainable in general practice. However, certain factors are commonly met with in the background of immigrants with these diseases. There are some who suffered disruption of their families, displacement, forced labour and other hardships, during and immediately after the last war. Some are stateless. As Weinberg points out, these people often experience language difficulties. It would not be surprising to find a high incidence of stress disease among such people. Some former displaced persons and refugees, and a few other immigrants, have escaped from an uncongenial environment in Europe to a better life here, at the cost of a lowered socio-economic status. Some have been disappointed in the conditions they have found here, having been misled or misinformed before they left Europe.

CASE VIII.—This man, now aged 47 years, was an accountant in Hungary before the war. He lived well and was able to support his wife in comfort. He was an army officer during the war, towards the end of which he and his wife and the three children they then had suffered the loss of their home and all their possessions, and were separated from the rest of their family, with whom they have lost all contact. He managed to get to Germany with his wife and children in 1945. Here, while living in the first of a series of camps, he developed the symptoms of a duodenal ulcer. After three years in camps the family migrated to Australia. There followed three more years in camps and hostels, and then, after he had secured work as a press operator in a large factory, they moved into the prefabricated weatherboard home where they now live. Not only does he work (when he is well enough), but so also does his wife, as an office cleaner; and husband, wife and four children crowd into the living room and one bedroom in order that the other two bedrooms may be rented to boarders. In addition to his ulcer, which is now of the order of severity often considered sufficient for operation, this man also has a very severe depression with some paranoidal tendencies. He has been impotent since just after he arrived in Australia. Although he has been here for ten years, he still speaks only haltingly in English.

CASE IX.—This man, now aged 63 years, was a business executive and an army reserve officer in Holland before the war. He had widespread cultural interests, and a large circle of intellectual friends. He spent most of the war as a prisoner of war in Dachau concentration camp, where he suffered a variety of illnesses, including the general effects of malnutrition, and active pulmonary tuberculosis. In 1950 he and his family—wife and four children—migrated to Australia. They paid their own passages. He came with the conviction that this country had more to offer his children than Holland. He was forced to sacrifice his business interests, and although he has secured a good job here, his position allows him less authority than he had in Holland. He has worked very hard indeed, as has his eldest daughter, mainly to ensure that the younger children will have a good education. He part-owns his home, which is roomy and well furnished with good quality furniture brought from Holland. He has had no outside interests at all since he came to Australia. He has found it difficult to make friends, though he and all the family speak perfect English. He misses the intellectual stimulation he had in Holland, and has come to depend entirely on his family, especially on his daughter, now aged 32 years, to satisfy his intellectual needs. Perhaps as a result, this girl is still single; she would like to marry and raise a family, but has devoted all her spare time to her father. Over the last three years this man has suffered increasingly frequent and severe bouts of depression, associated with a growing insight into his own advancing age and dependence upon his children. He is firm in his conviction that he did right in bringing his family to Australia, but it is impossible to say whether his spoken convictions conceal a subconscious doubt that may be growing stronger as the years pass. A rising blood pressure and the onset of angina of effort have impaired his vitality, and he misses the insurance he carried in Holland, which has lapsed, to be replaced by less secure sickness benefits in this country. In spite of the tubercle bacilli that may have been carried into the country by this man and his son, the family is a gifted one, capable of making a real contribution to the wealth of the nation in a way that would have been appreciated by Adam Smith. It is unfortunate that he, and perhaps other members of his family, may have to pay for this contribution in a way which jeopardizes their mental health.

The next case history demonstrates clearly a cause of neurosis that seems to be by no means uncommon in immigrants.

CASE X.—A female patient, aged 31 years, comes from a village in the beautiful mountain country in northern Yugoslavia. She has been in Australia for 18 months, having come out to join a man from the same village, to whom she had been betrothed since before he left five years earlier. She was married immediately after her arrival in Australia, and has been working for most of her time here as a cleaner and dresser of poultry. She and her husband live in one dark room of a small attached house, which is next door but one to a gasometer and a few hundred yards from a railway goods yard. Over the back fence is an iron foundry. She has been seen on several occasions over the last year with a variety of complaints, such as dyspepsia, insomnia, and nervousness which makes her drop and break things. She has also had a miscarriage. Unfortunately, interviews with this girl have to be conducted with the aid of her husband as interpreter, and psychotherapy through an interpreter is unlikely to be very successful. It is hard to escape the conclusion that much of her trouble is due to homesickness; it is a far cry from the mountains of Carinthia to her present dismal abode. She has been happier since the suggestion was made that her husband could get work in the country and they would thus be able to move from an obviously uncongenial environment. They plan to do this as soon as they have fulfilled their present obligations.

Inevitably in any planned programme of migration, unattached working men must form a considerable proportion of the people on the move. There are many social and economic reasons why this is so. It may be stated that all the great migrations of history have taken this form, and no doubt many variations of humanity have come about as a result of admixture which has occurred when these unattached men have married women resident in the countries to which they have travelled. In a small way, the same changes may be expected to occur as a result of the movement of people into Australia.

However, the presence of large numbers of single men in a country will create problems of its own. Some of these men suffered considerable hardship and often separation from all other members of their family during and after the last war. Many of these may be obliged, through age, language difficulties, physical unattractiveness or lack of capital, to resign themselves to celibacy. Some drifted or have been pushed from one labour camp to another for so long that they have become utterly dejected, and have lost all incentive to seek financial and marital stability. It is difficult to escape the conclusion that they would be bad bargains on the marital market in any case.

Others, notably the younger generation of single men from southern Europe, are sometimes betrothed to girls who will come out to join them after they have established themselves securely in this country. It would not be surprising to find these young men well adjusted to their life and situation, and in those cases in which a common language has made it possible to inquire, this has usually proved to be so.

To understand the small residue that do not adjust themselves well to life in the new country, motives behind migration must be considered. Many young men leave home because of a desire to better themselves, knowing that they will have far greater opportunities in the new country than in the country of origin. A spirit of adventure impels others. There will also be a small number of malcontents, rebels from society, criminals on the run, psychopaths and other assorted undesirables who may slip through the screen of selection in Europe. These will always tend to attract a disproportionate amount of attention to themselves, and it is their behaviour which is responsible for some of the hostility to immigrants which is evident in some sections of the community. Very few such people have been seen in this practice. One criminal

psychopath has caused a good deal of annoyance, and in the last five years perhaps half a dozen others have been seen.

Minor forms of maladjustment, usually due to some form of dissatisfaction with conditions encountered here, are less rare.

For the purposes of this discussion, venereal disease may be regarded as a consequence of maladjustment to the society in which the immigrant finds himself; in this practice it has been seen almost exclusively in immigrant men. When possible, these have been interviewed fairly closely (language difficulties sometimes make this impossible). In passing, it may be mentioned that venereal disease has never been seen in the young men from Greece or the Greek Islands. This may be due to chance, or to the absence of a reservoir of infection in the self-contained Greek community, but the impression has grown that it is at least partly because the upbringing of these young men has been so strict; in addition, many are betrothed to girls who will come out to join them when they are financially established. In this sense, the marital customs of the Greek world, with a high proportion of marriages arranged by the parents whose wishes are respected and obeyed, would seem to form a favourable background to successful assimilation.

CASE XI.—A male patient, aged 32 years, from Germany, in Australia seven years, a P.M.G. linesman, attended with a purulent urethral discharge which came on six days after intercourse with a prostitute picked up in a city nightclub. This was his third attack of venereal disease in three years. This man is just old enough to have been in camp at the end of the last war, ready to begin fighting. He spent most of the next few years in camps for displaced persons, and since coming to Australia he has continued in the same fashion, living almost all the time in P.M.G. camps. He has accumulated quite a large sum of money in savings, since he neither drinks heavily nor gambles. He has met few local girls, though he is a personable young man and should be attractive to women. There is probably some emotional immaturity in his outlook towards women, and at the few country dances he has attended with some of his mates from the camps he has mostly stayed on the sidelines with the other men, and has seldom taken the plunge onto the dance floor. In addition to the penicillin he was given for his gonorrhœa, he was given some simple psychotherapy, in the hope that it might help him adjust his personality to his present needs. It is too early to tell whether this has been successful.

Other case histories could be given to illustrate the point that immigration may profoundly affect mental health for a variety of reasons—reasons that may be simple or very complex. This is, of course, a matter that has engaged a lot of attention in recent years, and there is a large volume of medical and sociological literature on the general topic of immigration and its relation to mental health.

One final observation may be mentioned: evidence of child-parent conflict has been seen in several migrant families. Children will defy their parents or speak insolently to them in front of the doctor, calling them "ignorant" or "stupid" and so on. No attempt has been made to assess this phenomenon quantitatively. It is not unexpected, indeed its existence is predicted by psychologists who describe it as a manifestation of group prejudice, acquired by the children at school (Blake-Palmer, 1956). Out of this conflict between child and parent it is not impossible that delinquent behaviour may develop. This has been reported in the United States of America. In the next generation this may prove to be an important social problem in Australia.

The main point to be made is that impaired mental health in immigrants will be frequently seen in general practice, as indeed it will be seen in many walks of life; not all sufferers from mental ill-health will feel sufficiently distressed to visit a doctor. The important thing is that they should be recognized, and that action should be taken to deal with the problems they raise.

How may the general practitioner deal with these problems?

Listwan (1956, 1959) discusses this subject, and makes several points that have been found to apply in this practice. Kindness and sympathetic understanding will go a long way. An interest in the background of the immigrant and discussion of the ways in which the old life may have differed from the new, together with discussion of the problems of establishment in the new land, have been found on many occasions to be all that is needed in the way of psychotherapy. Migrants are always flattered to find a native Australian who has taken the trouble to learn a few words of their language—just as an Australian traveller on the continent of Europe will be grateful and flattered to be addressed in English.

Listwan describes other ways in which the individual and collective neuroses of immigrants may be broken down. Some of these are outside the competence of the general practitioner, except in so far as he may be able to educate other sections of the community.

Where the doctor is an important figure in the community, as he is in rural more than urban areas, much may be done in this way, particularly by education away from prejudice against migrants. There are several places in Australia where considerable rural communities of migrants have developed, such as the sugar-cane districts of northern Queensland. Borrie has described the prejudices that may be found in such areas. Prejudice may be transmitted into the second generation. The family doctor may be one individual who can help to break down this undesirable reaction by precept and example.

Summary.

Since the end of the second World War, the population structure of Australia has undergone a major transformation. The influx of well over 1,000,000 immigrants has raised many medical and social problems.

Some problems that may be encountered in general practice are described and discussed with illustrative case histories.

Diseases of interest that may occur in immigrants are: diseases present at entry into Australia; industrial and road accidents due to unfamiliarity with local speech and customs; tuberculosis, whether primary or reactivated; difficulties related to child-bearing; various forms of mental ill-health that may be causally related to immigration.

Therapeutic and preventive measures are briefly mentioned.

Acknowledgement.

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THE CONTRIBUTION OF ELECTROENCEPHALOGRAPHY TO THE KNOWLEDGE OF EPILEPSY.

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THIS paper presents a brief summary of the recent developments in electroencephalography and the influence they have had on the theory of epilepsy. It was thought it might prove of value to those who have not had access to all the relevent literature.

The clarification of the information available from the electroencephalogram tended to be delayed to the end of the last decade. Then two important disclosures were made. One was that seizures with the clinical and electroencephalogram features of *petit mal* and *grand mal* could be produced by the electrical stimulation of the mesial thalamus (Jasper *et alii*, 1947, 1949). The other was the realization by Gibbs, Gibbs and Fuster (1947) that the square-topped waves which they had found generalized over the cortex in their electroencephalogram records from epileptics who suffered from "psychomotor" seizures were really an artefact due to a localized regular spike discharge in the temporal lobe. As they were employing unipolar recording and using the ear as a supposedly inactive reference electrode, the regular spike was being picked up by this reference electrode and so appearing inverted in all the leads as a wave pattern.

These two disclosures allowed the distinction to be made between seizures originating in the cortex and so being associated with a local cortical discharge in the electroencephalogram, and those originating in subcortical structures and so being associated with a bilateral and generalized abnormality. This distinction made the clinical features much more intelligible, as well as proving valuable diagnostically and therapeutically, so that the previous attempts to find specific patterns to match the clinical appearance of the seizure, i.e., in terms of *grand mal*, *petit mal* and "equivalents", were quickly abandoned and a classification adopted based on this distinction. Actually, this marked a return to the attitude of Hughlings Jackson:

> The first question in my mind is not "is it a case of epilepsy?" but "where is the lesion permitting occasional excessive discharge?".

The value diagnostically of this classification is apparent from the fact that focal cortical epilepsy is due to local structural change, whereas subcortical epilepsy is associated with diffuse cerebral damage or none at all. Its importance therapeutically is determined by the recognition that the anticonvulsants in common use tend to act differently on the different types. For example, "Mysoline" and phenobarbitone are generally effective in both cortical and subcortical epilepsy, whereas "Dilantin" is effective only in cortical epilepsy and the diones only in true subcortical *petit mal*.

Seizure Types.

The clinical features which accompany a subcortical discharge are, of course, the *grand mal* (not including the "aura") or the *petit mal*, which may be a clouding or a loss of consciousness with or without myoclonic jerks, but not preceded by an aura and characterized by the immediate return of clear consciousness. The ictal electroencephalogram in these conditions differs markedly, of course. *Grand mal* is accompanied by spikes, the frequency of which seems to determine the accompanying motor features. They are rapid at the commencement, preventing the muscles from relaxing, thus producing the tonic state. As they increase in amplitude and slow in frequency, a point is reached where the muscles can relax in between the spikes—the clonic stage. Finally, there is temporary cortical inactivity associated with the state of temporary coma. *Petit mal* characteristically accompanies the spike and wave pattern commencing at 3·0 to 3·5 cycles per second and slowing to about 2·0 cycles per second. However, both these disturbances are bilaterally symmetrical and generalized, as are the inter-ictal electroencephalographic abnormalities when present. Unlike the ictal electroencephalogram patterns, these inter-ictal features are unrelated to the seizure type.

The cortical seizure, depending on its locus of origin, may be accompanied by any of a wide variety of phenomena, all of which are included in the term "the epileptic aura". Actually, this term has several disadvantages, suggesting as it does that it includes only symptoms experienced and remembered by the patient and that it precedes the attack, whereas it is actually the beginning of, or in fact may be, the entire attack. Also, it is often classified as part of a *grand mal*, which as stated above is an entirely separate event and may or may not follow a cortical seizure.

Depending on the area of origin of the focal cortical discharge, any type of motor, sensory of autonomic signs or symptoms may appear, including psychical disturbances —especially with lesions in the temporal lobe. These psychical seizures vary from lapses clinically identical with those of *petit mal* to complex illusional or hallucinatory states. Rarely a cortical seizure can last a considerable time, when it is termed epilepsia partialis continua, but usually it does not last more than one minute.

As stated above, the cortical seizure may or may not be followed by a *grand mal* — usually explained when it appears as due to a spread of excitation to the subcortical mechanism. That such a spread occurs within the cortex is evident in the march of Jacksonian or similar sensory attacks. However, whether a *grand mal* occurs or not, a cortical seizure may be followed by a period of confusion or loss of function, e.g., paresis or aphasia. This, like the seizure symptoms, is of localizing value.

The electroencephalogram during a clinical attack is naturally not often recorded. Most commonly it is described as showing a spike discharge increasing in voltage and decreasing in frequency as in the *grand mal*, but of course localized to the affected cortical area. The inter-ictal resting electroencephalogram is commonly abnormal, again the abnormality being localized to the affected area and usually consisting of periodic spikes or spikes and waves, or at times of runs of slow waves, tending to be more regular than slow waves seen with local cerebral gross organic pathological conditions. However, with this less specific electroencephalogram, or indeed when the electroencephalogram is normal, sleep, natural or induced, is generally used and often produces more specific electroencephalographic abnormalities.

One difficulty more commonly encountered in children with cortical and especially with temporal lobe epilepsy is that the inter-ictal electroencephalogram may show a bilateral generalized and fairly symmetrical spike and wave discharge, though often not of the typical 2·5 to 3·5 cycles per second frequency and classical pattern. This is thought of as a secondary subcortical discharge, and indeed, if carefully examined, the beginning of the discharge may occur first on the affected side. Here sleep activation and possibly the use of sphenoidal electrodes may demonstrate the focus, or routine electroencephalograms taken a few years later may indicate it.

As well as the pure cortical and subcortical discharges, at times electroencephalograms may show features of both. Generally, multiple inconstant cortical foci are seen mixed with subcortical discharges on a background rhythm which is severely abnormal, showing dominant irregular slow activity. This type of epilepsy is generally due to diffuse cerebral atrophy, and is classified as diffuse dysrhythmia. Usually, even when adequately treated with anticonvulsants—that is, with a maximum of effective drugs being employed in maximum effective doses—these patients' seizures cannot be completely controlled. In this matter of anticonvulsant dosage it is generally agreed that a considerable time must be spent working out the maximum effective dose of each drug, and there should be no hesitation in maintaining it just short of the toxic level—a level which has to be discovered independently for each patient.

Concepts Now Being Discarded.

Prior to the increased knowledge of epilepsy given by the electroencephalogram, concepts have been introduced which have not been completely accurate. Possibly the most harmful is that of the "epileptic equivalent", of which Penfield (1954) has said it is "wiser to discard the expression". As he sensibly points out, the patient either has true seizures or some other condition altogether.

However, less easy to discard are such concepts as that of "automatism". This seems to be the last refuge of the tendency to regard epilepsy as a divine illness. The term suggests that epilepsy gives the individual power to carry out, without knowledge or intent, behaviour which virtually knows no limits. As, along with ictal behaviour, any behaviour occurring in the post-ictal confusional state is included in the term, this all-inclusiveness is understandable. However, why should behaviour in the confusional state following a cortical seizure or *grand mal* be labelled automatic, as opposed to that occurring say in hypoglycæmic, post-traumatic or alcoholic confusion? Also, many episodes of aggression occurring in psychopaths and many hysterical fugues can be considered epileptic on the basis of this concept, even though there may be no history of their ever having followed a definite disturbance of consciousness or having occurred in a mental state that was clearly abnormal and not merely an emotional outburst.

What has added to the confusion is that lesions of the temporal lobe, as well as causing epilepsy, may be associated with a severe degree of aggressive or paranoid behaviour. However, this is not justification for regarding episodes of aggression occurring in temporal lobe epileptics as manifestations of epilepsy. Similar episodes may occur in patients with temporal lobe lesions who have no epileptic seizures. Some of these patients may even show temporal lobe spikes without clinical manifestations of epilepsy (Hill, 1952). A further difficulty is that a high percentage of psychopaths tend to have abnormal electroencephalograms, apart from those just mentioned. However, these records would not usually be confused with those of epileptics. Mainly they show signs of immaturity compared with those of normal controls of the same age. This is more evident after the age of 25 years, when the records of the normal rarely show much evidence of these signs.

It would seem wiser at the present juncture to err, if at all, on the side of conservatism, and if the term automatism is to be used, to restrict it to the simple behaviour that can continue through a *petit mal* and to the masticatory, the simple fumbling and pushing or pulling movements or the mumbling of a few words that are seen in focal seizures, which are mainly temporal or frontal lobe in origin. When seen in this ictal condition, the patient appears inwardly preoccupied and cannot direct his attention on to you, whereas in the post-epileptic confusion, he can obviously direct his attention on to you, but it is a very coarse, easily distractable, sort of attention.

Psychomotor Epilepsy.

Another term often encountered in the past which is gradually being replaced is that of "psychomotor epilepsy". It was introduced by Gibbs in his attempt to find clinical entities to match generalized electroencephalogram patterns. In this case, as was pointed out in the introduction, the generalized pattern he described was an artefact actually produced by spikes in the temporal lobe. Hence, most of these types of attack are now referred to as temporal lobe epilepsy. The term "psychomotor" stressed the two most interesting aspects of temporal lobe epileptic seizures—that the seizure may be "psychic" and that it can include the type of ictal automatisms mentioned above as well as post-ictal confusional behaviour. The psychic seizure is one in which complex mental phenomena occur—illusions, e.g., *déjà vu* or *jamais vu* states, complex hallucinations, which can be auditory, visual, olfactory or gustatory, emotional states, usually of fear, or forced thinking. Of course, temporal seizures very commonly show none of these interesting features, but merely a simple aura, often a rising epigastric sensation, possibly followed by a brief lapse or a *grand mal*.

The Epileptic Personality.

Another concept that is much discussed is that of an epileptic personality. It is now generally thought that this is seen, apart from that due to mental deficiency or cerebral degeneration, mainly in epileptics with temporal lobe lesions. Hill has recently stated that half of these have personality disorders (he also considers that probably one-third of all epileptics have temporal lobe lesions). The type of personality disorder is that usually described—namely, a tendency to irritability, jealousy, intolerance, suspicion, paranoia, unreasonableness, moral righteousness and aggressiveness. Episodes of frank paranoid delusional states are common in these people. It is commonly stated that when epileptic seizures are controlled by anticonvulsants, the patient's behaviour is worsened. This can happen, but it seems very much the exception rather than the rule. When it does occur it seems to be in this group of epileptics.

Value of the Electroencephalogram.

Having discussed the value of the electroencephalogram in the theory of epilepsy, I shall conclude by summarizing present views on its value in the individual case.

It is generally accepted that it should be employed to investigate and localize the epileptic lesion, not to diagnose the presence or absence of epileptic manifestations in the patient. A single normal electroencephalogram should never be regarded as proof of the absence of epilepsy. In fact, this is not uncommon in generalized epilepsy, especially if the attacks are infrequent, or in temporal lobe epilepsy. The sleeping electroencephalogram is more likely to be abnormal, especially in the latter case.

The diagnosis of epilepsy is a clinical one, but its localization is frequently impossible or inadequate without electroencephalographic investigation.

Summary.

Knowledge derived from the electroencephalogram is largely responsible for the present classification of epilepsy into cortical and subcortical seizures. This classification has proved valuable diagnostically and therapeutically. A description of the seizure types and the associated electroencephalogram disturbances is given. Certain concepts, notably that of the "epileptic equivalent", are criticized, and it is suggested that the use of the term "automatism" be restricted. "Psychomotor epilepsy" and the epileptic personality are discussed, and a summary of the use of the electroencephalogram in an individual case is given.

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UNUSUAL EARLY SYMPTOMATOLOGY OF PRIMARY MALIGNANT TESTICULAR NEOPLASMS.[1]

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THOUGH the testicle is a relatively superficial organ, its neoplastic lesions, like those of the breast, may be extremely difficult to diagnose, especially if they present in an unusual or a bizarre form. This is unfortunate, as early diagnosis is important because metastases can occur so quickly that any equivocal lesion of the testis at any age from infancy to senescence should be suspected as being a neoplasm requiring early exploration.

In a short personal series of 20 cases of primary testicular neoplasms spread over the last 20 years, 11 presented initially in the classic manner as either a painless or an aching small swelling present a varying length of time before operation. Many of these were accompanied by a hydrocele, which in no instance was conspicuous or large. However, its presence at operation showed that the growth was involving the visceral layer of the tunica vaginalis, whereas its absence showed that the growth was still in the centre of the testis.

The remaining nine cases presented in an atypical fashion.

Initial Symptoms Due to Metastases from an Unobtrusive Primary Testicular Neoplasm.

In each of two instances there was an unobtrusive primary growth in a testicle clinically normal as far as both palpation and sensation were concerned. Presenting symptoms arose from the secondary metastases.

CASE I.—The patient had a pseudo-inflammatory mass in the skin of the neck, which was originally thought by his doctor to be an infected sebaceous cyst. Exploration and pathological examination revealed the structure of a seminoma.

CASE II.—A non-productive cough drew attention to the patient's lungs, in which radiography revealed numerous metastatic shadows.

Both of these patients quickly succumbed to widespread secondary metastases. Subsequent autopsy in each instance revealed a minute seminoma in the centre of an outwardly normal testis.

It is of more than passing interest to note that in 100 cases of primary testicular neoplasm reported by Stephen (1958), 30 had metastases before operation.

Lack of Sensation as the Only Initial Sign.

In two instances in this series testicular sensation was wholly absent. This was the only abnormal sign found in these two patients, who complained only of aching in the affected organ. In the other cases in this series, testicular sensation was either completely normal in the case of small central neoplasms or absent only where the tumour touched the visceral layer of the tunica vaginalis.

CASE III.—In the first case of completely absent testicular sensation, the affected testicle ached. Palpation revealed that though the testicle felt normal in size and shape, even when compared with that of the opposite side, it lacked testicular sensation. Orchidectomy revealed that almost all the testis was replaced by seminoma.

CASE IV.—In the second case, this lack of sensation in an aching testicle gave the clue to a small seminoma. The patient had a single testis, the other having been removed surgically for injury some years previously. The remaining testis began to ache, and examination showed a testis apparently normal in size and shape, though, of course, there was no normal one for comparison. However, the lack of sensation led to exploration, which revealed a seminoma.

Whilst on the matter of the single testis, Stephen (1958) points out that now that surgery and radiotherapy lead to prolonged survivals after removal of a testicle for neoplasm, neoplasms are showing up with more frequency in the remaining testicle. This occurred in three of his 100 cases. In one case the original neoplasm was a seminoma, but the later one in the other testis was a teratoma.

Initial Symptoms after Injury.

CASE V.—A blow on the testis first drew the surgeon's attention to one young army recruit. The testis, about which the patient had never noticed anything unusual previously, was kicked at football, resulting in a large but relatively painless hæmatocele and hæmatoma of the testis. The patient's medical officer was impressed by the absence of pain and immediately sought surgical opinion. Exploration revealed a hæmatocele surrounding a split testis, the surface of which showed the white homogenous structure of a seminoma.

[1]Read at the B.M.A. clinical meeting at the Royal North Shore Hospital of Sydney in April, 1959.

Initial Symptoms Due to Spontaneous Hæmorrhage.

CASE VI.—One such case presented as the so-called "pseudo-inflammatory type" in a young man who had for six months suffered recurrent attacks of severe pain with swelling of the affected testicle, which each time subsided on antibiotic therapy. Each attack was accompanied by a rise in temperature. In the last attack, examination revealed an enlarged tender testis and epididymis without a hydrocele, not unlike an acute virus epididymo-orchitis. Operation, however, revealed a testis enlarged by seminoma. There were numerous hæmorrhages both in the growth and in the normal testicular tissue surrounding it.

CASE VII.—Sudden massive spontaneous hæmorrhage occurred in another young adult, leading to a very large and agonizingly painful testicle, which was first diagnosed as a torsion because it was so painful and shocking. However, it was realized that this diagnosis was vitiated by the large size of the scrotal mass. Operation revealed some testicular tissue, infiltrated with blood but otherwise normal, surrounding a pseudocyst full of blood clot, in which floated a necrotic mass later shown to be a seminoma, some cells of which were also in the wall of the cyst. The hæmorrhage had also burst the testis, causing a hæmatocele.

Initial Symptoms Due to Torsion.

CASE VIII.—Acute torsion of the extravaginal cord in a young man led to operation, which revealed an unsuspected small teratoma in a necrotic testicle.

Confusing Initial Clinical Picture.

CASE IX.—A young man's children developed acute epidemic parotitis. Shortly afterwards, to his horror, he found that his left testicle had become smaller than his right. Though he had never had parotitis or lost any sensation from the allegedly atrophic testicle, the family history of mumps was a sufficient "red herring" for his doctor to be misled into giving testosterone injections until it was realized that the left testicle was not so much smaller than the right, but that the right was larger than the left. This was true, as the right testicle contained a seminoma. However, six months had been wasted before operation.

Discussion.

The above-mentioned unusual early symptoms in this short series by no means exhaust the list of such symptoms.

In this series, for instance, there were no patients with gynæcomastia. In Stephen's 100 cases only two showed this sign. It is uncommon; in fact in 218 cases of gynæcomastia reported by Sirtori and Veronisi (1957) from Italy, only four were due to testicular neoplasms.

In my series the neoplasm did not occur in cases of mal-descent or non-descent. In Stephen's (1958) series, only four occurred in mal-descended testes and none were intraabdominal. Actually six of his patients had mal-descended testicles, but in two cases the neoplasm occurred in the contralateral normally placed scrotal testis. In those four cases in which the mal-descended testis was affected, in two it occurred in the untreated testicle at the external ring and in the other two it occurred some years after an orchidopexy had successfully placed the testicle in the scrotum, where it had remained.

It is said that a testis which is the subject of a failed orchidopexy should always be removed as it is particularly liable to neoplasm. This also applies to the undescended testicle wrongly replaced in the abdominal cavity instead of being excised.

Conclusion.

Enough has been said to show that the early diagnosis of testicular neoplasm may be difficult, because of equivocal symptomatology; but it is most important because of its effect on prognosis. Any doubtful testicular lesion in any age group should be explored as it may be a neoplasm.

Summary.

A short series of 20 cases of testicular neoplasms is presented, in nine of which the early symptomatology was unusual and equivocal.

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RUPTURE OF THE UTERUS.

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RUPTURE of the uterus is one of the gravest complications of pregnancy and labour. Its incidence in hospital practice shows wide variation, as a review of the literature will show. This variation may be thought to reflect the standard of obstetrical care, but it depends on additional factors such as the demography of the area which a hospital serves, the criteria for hospital confinement, the proportion of unbooked cases and the accuracy of diagnosis of uterine rupture.

The ætiology of the condition has been altering slowly, for there has been a steady decline in the numbers resulting from obstetrical trauma, and an apparent rise in the number of ruptures following the increased use of Cæsarean section. The change from classical to lower segment Cæsarean section and the control of puerperal pelvic infection have made this increase relative rather than absolute.

Jean Lebar of Mouillerou in 1766 first attempted the repair of a ruptured uterus, although in the sixth century Aëtius of Amida was aware of the risk of tearing the uterus when delivery was difficult.

PRESENT STUDY.

For the present study the case records of the Obstetrical Units at the Royal Women's Hospital, Melbourne, were examined for the seventeen-year period 1939 to 1956, and 50 cases of uterine rupture were abstracted. This material was then analysed to relate the clinical findings with the causative factors and the pathology. The management of a pregnant patient with a Cæsarean section scar is considered.

INCIDENCE.

The incidence of uterine rupture in patients whose antenatal care was undertaken at the Royal Women's Hospital was one in 2710. Lavery (1955) quoted an incidence in the Bantu native of one in 137, whilst the incidence in other series varied from one in 1000 to one in 3000. Some of the factors affecting the incidence of this complication have been mentioned. In addition, the accuracy of case-recording and analysis may be important, as in a prospective study covering three years Pedowitz and Schwartz (1957) discovered 48 cases of uterine rupture, which was in contrast to the three cases recorded in the preceding 20 years.

ÆTIOLOGY.

The average age of the patients, 34 years, was considerably higher than that of the majority of the women confined in the hospital, 26 years. There were four primigravidæ and seven patients who had had five or more previous pregnancies. A complete list of the causes can be made by considering the physiological and pathological factors resulting in either a weakening of the wall of the uterus or an increase in its internal pressure. As simplicity is the most important consideration in any classification the cases have been divided into spontaneous and traumatic rupture and scar rupture (Table I).

Spontaneous Rupture of the Uterus.

Obstructed Labour.

In four cases, uterine rupture followed obstructed labour due to cephalo-pelvic disproportion. Three of the patients were multigravidæ, who were in the second stage of labour longer than four hours. The other patient was a primigravida in the fortieth week of gestation, who laboured for 48 hours with a vertex presenting, the head not engaged and the membranes ruptured. These cases occurred in the first five-year period of this review. An unrecognized transverse fœtal lie was the cause of obstructed labour and subsequent uterine rupture in a further three cases.

Uterine Abnormality.

Abnormality of the uterus may have contributed to the cause of its rupture in four cases.

In one patient, the first pregnancy resulted in a complete abortion. During the patient's second pregnancy the uterus ruptured before the onset of labour, at the thirty-ninth week of gestation. The tear was situated vertically in the midline of the anterior wall of the uterus, and there was extensive trophoblastic infiltration of the complete thickness of the myometrium at the site of the rupture.

This case was an example of the rare condition placenta percreta (Cuthbert, 1956).

In two multigravidæ, uterine rupture occurred through areas of adenomyosis. Other ætiological factors were present in each case. In one, rupture followed the intramuscular injection of pituitrin to induce labour, and in the other, rupture occurred after ten hours of labour, when the fœtal lie was transverse.

TABLE I.

Ætiology of Rupture of the Uterus.

Cause.	Number of Cases.
Spontaneous rupture:	
Obstructed labour	7
Uterine abnormality	4
Idiopathic rupture	8
Traumatic rupture:	
Difficult forceps delivery	5
Manual removal of the placenta	3
Internal version and breech extraction	2
Destructive operation	1
Motor-car accident	1
Scar rupture:	
Classical Cæsarean section	14
Lower segment Cæsarean section	3
Myomectomy	1
Tubal implant	1

The remaining patient had an intrapartum pelvic infection, and histological examination of the ruptured uterus revealed infected thrombi, œdema and inflammation of the myometrium.

Although Potter (1930) obtained microscopic evidence of abnormality of muscle fibres in 50% of cases of uterine rupture, the ætiological significance of these findings has not been proved.

Idiopathic Rupture.

There were eight cases in which there was no obvious cause for the uterine rupture, and the clinical features of this group are shown in Table II. Severe cervical lacerations may follow a precipitate labour in the multiparous patient, when it is probable that the uterus forces the presenting part through an incompletely dilated cervix. The same mechanism may result in uterine rupture. The occurrence of a short labour, a rupture extending into the cervix and the frequency of diagnosis after delivery support this hypothesis. In addition, Murphy (1947) and Jeffcoate (1950) consider that the increased tendency of the multiparous uterus to rupture in labour is due to its stronger action. On the other hand, Munro Kerr and Chassar Moir (1956) suggest that parity weakens the uterus, and thus accounts for its increased tendency to rupture.

Traumatic Rupture of the Uterus.

In the five forceps deliveries resulting in uterine rupture, either the fœtal head was not engaged or the cervix was not fully dilated at the time of delivery.

Manual removal of the placenta caused three uterine ruptures. In one a placenta accreta was present, in another a placenta prævia and myomectomy scar were present, and in the third a previous manual removal had been carried out and the placenta was adherent.

In two cases the fœtal lie was transverse, and internal version and breech extraction precipitated the uterine rupture. When the fœtal lie is transverse, internal version may be performed with safety if the cervix is fully dilated, the membranes are intact and the pelvis is adequate (Wood and Forster, 1959).

The destructive operation was an evisceration attempted on a dead fœtus lying transversely. If internal version or decapitation is not possible in these circumstances, then Cæsarean section should be performed (Munro Kerr and Chassar Moir, 1956). Previous curettage has been suggested by Mair (1950) as a predisposing factor in uterine rupture. In this series eight of the 50 patients had a history of a previous miscarriage, but this rate is similar to that of patients delivered in the hospital.

TABLE II.

Clinical Features of Eight Cases of Idiopathic Uterine Rupture.

Patient's Age. (Years.)	Parity.	Duration of Labour. (Hours.)	Description of Uterine Tear.	Time of Diagnosis.	Previous Curettage.
41	8	2	Cervix to fundus.	After delivery.	—
39	7	3	Cervix to fundus.	During delivery.	—
40	3	2	Cervix to upper segment.	After delivery.	1 dilatation and curettage
37	7	2	Cervix to upper segment.	After delivery.	—
44	4	3	Lower to upper segment.	After delivery.	1 dilatation and curettage
33	2	4	Vagina to fundus.	After delivery.	—
31	2	4	Cervix to fundus.	After delivery.	—
37	3	3	Cervix to upper segment.	During delivery.	—

Uterine Scar Rupture.

Classical Cæsarean Section Scars.

The clinical onset was acute in all cases, and the uterine rupture complete in 13. There were no maternal deaths, and four of 16 babies survived. Half the ruptures occurred prior to the onset of labour—six between the thirty-sixth and fortieth weeks of gestation and one in the twenty-sixth week. In two cases of twin pregnancy, rupture occurred in the middle trimester. Two or more Cæsarean sections had been performed in three cases, and in one of these a classical and a lower segment scar intersected at the site of uterine rupture. In five cases the placenta was attached at the site of the scar rupture.

Lower Segment Cæsarean Section Scars.

There were only three cases of lower segment scar rupture, and there were no fœtal or maternal deaths.

The first patient had two previous lower segment Cæsarean sections, and a further Cæsarean section was performed, as labour had ceased to progress after 21 hours. There were no symptoms or signs of a ruptured uterus. At operation, a two-inch defect in the uterine scar was found, and the fœtus and placenta were still in the uterus. The bladder was torn during a difficult delivery of the fœtal head. The uterus and bladder were repaired, and the patient made an uneventful recovery.

In the second case, the brow of the fœtus presented and descended to a low pelvic station after a ten-hour labour. A dehiscence of the scar was suspected on abdominal palpation; but as there were no signs of uterine rupture the head was flexed to convert the brow to a vertex presentation, and delivery was completed with forceps. Manual exploration of the uterine cavity revealed a complete tear, and subsequently subtotal hysterectomy was performed.

The third patient had been in labour for nine hours when there was a small loss of blood per vaginam, and the uterine contractions diminished. Rupture of the uterus was diagnosed, and laparotomy showed dehiscence of the scar, intact membranes and the fœtus within the uterus. The baby was delivered, the uterine scar excised and the uterus repaired.

Other Scars.

Manual removal of a placenta prævia precipitated rupture of the uterus in a patient with a myomectomy scar. The anatomical relation of the uterine tear to the myomectomy scar was not determined.

Another patient had a bilateral tubal implant performed for infertility. In her first pregnancy a dead fœtus was delivered by the breech after a short labour in the thirty-second week of gestation. The patient became shocked, and manual exploration of the uterus revealed a half-inch tear at the site of the tubal implant. A subtotal hysterectomy was performed, and microscopic examination of the uterus confirmed that the placenta was attached to the uterus at the site of the tubal implant.

Diagnosis.

Rupture of the uterus may become manifest during pregnancy, labour and the puerperium. The relation of the three phases of childbearing to the cause of the rupture is shown in Table III. The assessment of symptoms from the case records was not considered to be complete.

TABLE III.

Relationship of the Time of Diagnosis to the Cause of Uterine Rupture.

Cause.	Number of Cases.
Pregnancy:	
Classical Cæsarean section scar	7
Uterine abnormality	2
Motor-car accident	1
Labour:	
Obstructed labour	7
Classical Cæsarean section scar	7
Idiopathic rupture	2
Lower segment Cæsarean section scar	2
Uterine abnormality	2
Puerperium:	
Traumatic delivery	11
Idiopathic rupture	6
Uterine scar	3

During Pregnancy.

The diagnosis was made during pregnancy in ten cases. The commonest symptoms were abdominal and shoulder tip pain. Vaginal hæmorrhage occurred in four cases. Shock and abdominal tenderness were more reliable signs than the absence of the fœtal heart; the fœtus was felt separate to the uterus in three cases.

During Labour.

In 20 cases the diagnosis was made during labour. In most cases the uterine contractions ceased and the patients became shocked. In two cases the membranes were intact at the time of uterine rupture. Hæmaturia was reported on two occasions. Lavery considers this an important sign, and perhaps its significance is sometimes overlooked.

Small (1953) has emphasized the fact that the onset of rupture of the uterus was not often dramatic. This statement was true for the lower segment scar rupture. Its insidious onset may be indicated by the weakening of contractions, vaginal hæmorrhage, fœtal distress, a rising maternal pulse rate and failure of labour to progress. The sudden onset of tenderness or a palpable gap in the scar may aid in the diagnosis. The rupture of a lower segment scar may be silent, and dehiscence of the scar may be found at a second Cæsarean section (Pedowitz and Schwartz, 1957).

During the Puerperium.

The diagnosis was made during the puerperium in 20 cases. Post-partum hæmorrhage and shock were the usual signs of a ruptured uterus. In five cases the diagnosis would have been made earlier if manual exploration of the uterus was carried out as a routine procedure after any difficult vaginal delivery, after precipitate labour or after vaginal delivery in the presence of a uterine scar.

Treatment.

The death of several patients prior to the commencement of treatment (Table IV) emphasizes the necessity for speed in the management of these cases. When the patient is moribund, multiple blood or plasma transfusions are begun, and if the patient's condition does not improve laparotomy should be performed immediately. A subtotal hysterectomy is carried out when the uterine tear is large and irregular or extends into the broad ligament. If the tear is small and accessible, it may be sutured.

If the patient is not shocked, parity as well as the type of uterine tear will influence the choice of operation. If the uterus has to be removed in these circumstances, total hysterectomy may be performed to avoid the risk of occurrence of carcinoma in the cervical stump. Dyer (1957) suggests that total Cæsarean hysterectomy does not increase the operative time or provoke bleeding. This statement is not generally accepted.

TABLE IV.

Treatment of Patients with Rupture of the Uterus.[1]

Treatment.	Number of Cases.
Subtotal hysterectomy	32
Repair of uterus..	10
Conservative (non-operative)	3

[1] Five patients died before treatment.

Prognosis.

There were seven maternal deaths. Three followed spontaneous uterine rupture in a precipitate labour, two followed obstructed labour, one followed a "failed forceps" delivery, and one resulted from a motor-car accident. There were 37 fœtal deaths.

DISCUSSION.

The risk of uterine rupture is the main factor to be considered in determining the management of a pregnant patient with a Cæsarean section scar. The number of cases of Cæsarean section scar rupture in the present series was insufficient for any conclusions to be drawn concerning the influence on the strength of the scar of factors such as the site of placental attachment, previous post-section morbidity and twin pregnancy. An increased incidence of uterine rupture when the placenta was attached at the site of the scar led Morrison and Douglas (1945) and Wilson (1951) to perform X-ray placentography before term on all pregnant patients with a uterine scar. Wilson was also able to show a correlation between previous post-section morbidity and the incidence of uterine scar rupture. From a study of 16 cases of Cæsarean section scar rupture, Feeney and Barry (1955) concluded that the risk of this occurrence was increased by each vaginal delivery, high parity, twins, hydramnios, a large fœtus and implantation of the placenta over the scar. Although there are theoretical reasons to support their conclusions, they were based on a small number of cases. Pedowitz and Schwartz (1957) carried out a prospective study of Cæsarean section scars for three years, and concluded that their behaviour in a future pregnancy could not be predicted from the knowledge of the site of placental attachment or previous post-section morbidity. A more comprehensive survey to determine the factors influencing the strength of a uterine scar still needs to be made.

The behaviour of the classical and transverse lower uterine segment scars must also be considered. At the Royal Women's Hospital, Melbourne, a study of 881 pregnancies following a previous Cæsarean section showed the incidence of classical Cæsarean section scar rupture to be 5% (5 cases out of 95) and lower segment scar rupture to be 0·4% (3 cases out of 738). In a collected series Eames (1953) calculated that the risk of death to a patient who went into labour, when the indication for the previous Cæsarean section was no longer present, was 0·05% for a classical scar and 0·01% for a lower segment scar. Both these figures were less than the risk of an elective Cæsarean section, 0·08%. In Eames' series, the incidence of uterine rupture in patients going into labour with a lower segment scar was 1·3% and with a classical scar 2·6%. Because the incidence of uterine rupture was different at the Royal Women's Hospital, and because the maternal morbidity and fœtal mortality of uterine rupture have been ignored, one cannot accept Eames' conclusion that if the indication for the previous Cæsarean section was no longer present, a vaginal delivery could be anticipated for patients with both types of Cæsarean scar. Lawrence (1953) was even more conservative and suggested that a trial of labour might be safe after a previous Cæsarean section for a contracted pelvis. Gordon (1957), and Johnston and Morgan (1956), on the basis of extremely low mortality rates for Cæsarean section—0·01% and 0·15% respectively—follow the rule of "once a Cæsarean, always a Cæsarean".

It is impossible to reach a valid statistical conclusion as to the indications for repeating a Cæsarean section. In view of the multiple factors which have to be considered, it is not surprising that different methods of treatment are employed. As the incidence of classical Cæsarean section scar rupture was 5% at the Royal Women's Hospital, Melbourne, the present policy—of repeating all classical Cæsarean sections, and a lower segment Cæsarean section only when the indication was recurrent—remains acceptable.

At the Royal Women's Hospital, Melbourne, most patients are sterilized at the time of the third Cæsarean section. A large number of factors must be considered in reaching a conclusion as to the indication for sterilization: the maternal mortality of the second, third or fourth Cæsarean section; the risk of uterine rupture in pregnancy after successive Cæsarean sections; the maternal morbidity and late effects of section compared with those of a normal delivery; and the obstetrical history, general health and mental attitude of the patient. McNally and Fitzpatrick (1956) reported the risk of uterine rupture in 130 patients with a history of four or more Cæsarean sections as 1·5%. The lower uterine segment can be assessed by the surgeon at each Cæsarean section, and when the previous scar can be excised and the uterus repaired, the risk of uterine rupture in a subsequent pregnancy may not be increased. The desire for another child may be more important to a woman than the risk of another Cæsarean section. Probably with this thought in mind, Barnes and Zuspan (1958) studied the patient's reaction to puerperal surgical sterilization, and found that nine of 65 Cæsarean section patients unequivocally regretted having tube resection. Considerable care should be taken in deciding when sterilization is necessary after multiple Cæsarean sections.

The management of a patient in a pregnancy after a previous uterine rupture requires some consideration. The risk of a further uterine rupture cannot be accurately assessed. Although Mikhine collected 13 cases of repeated uterine rupture in 1902, case reports in recent years are not common (Lyons, 1950; Pettit, 1943). Linington (1956) suggests that if the tear or scar rupture was similar to the lower segment incision, then it should stand further vaginal deliveries.

SUMMARY AND CONCLUSIONS.

1. A review of 50 cases of rupture of the uterus treated at the Royal Women's Hospital, Melbourne, between 1939 and 1956 has been made.

2. The true incidence of uterine rupture is difficult to determine.

3. The causes of uterine rupture have been considered. Idiopathic rupture of the multiparous uterus probably results from its overaction.

4. The diagnosis of rupture of a lower segment Cæsarean section scar may be difficult, and its varied clinical features have been described.

5. Manual exploration of the uterus should be carried out after a difficult vaginal delivery, a precipitate labour or a vaginal delivery in the presence of a uterine scar.

6. It is impossible to reach a valid statistical conclusion as to the indications for repeating a Cæsarean section. The present policy of repeating all classical Cæsarean sections, and a lower segment section only when the indication is recurrent, remains acceptable.

7. Considerable care should be taken in deciding when sterilization is necessary after multiple Cæsarean sections.

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THE BRIDGE FROM TREATMENT TO EMPLOYMENT.

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Although all the material for the bridge between treatment and employment may be placed most precisely in position, faith and good will are still needed to make the journey from one end to the other. Attempts have been made to drive the patients across and to force open the gates of employment. These methods must always be doomed to failure.

The road to success with the employment of the disabled is in the training of personnel at both ends of the bridge; in establishing an understanding both of the potentialities of treatment and of the scope of employment. Legislation can be no substitute for education.

—GRIFFITHS (1952).

IN Britain, a review (Southworth, 1957) of the work of the statutory scheme for placement of the handicapped concluded: "We in the Ministry of Labour are by no means complacent about the Disablement Resettlement Officer (D.R.O.) service . . ."

The Piercy Committee[1] in Britain recommended that hospital boards should give attention to that which would "secure a purposeful graduated programme of activity designed to restore full activity and to re-orientate the patient's outlook from that of an invalid to that of a responsible worker" (Editorial Article, 1956). The Piercy Committee further stressed the need for close consultation between the doctor and the disablement resettlement officer (D.R.O.), and recommended that for specially difficult cases resettlement clinics should be established at every major hospital. Thirdly, the Piercy Committee recommended that as an experiment, industrial rehabilitation units should be combined with the rehabilitation services of suitable hospitals, to make the former units more effective and more economical.

Vocational Counselling.

Oddly, the Piercy Committee did not appear to note a need, on the bridge between treatment and employment, for anybody between the almoner at one end and the D.R.O. at the other. Complementarily the Piercy Committee found that arrangements regarding vocational training were satisfactory, and had no recommendations to make. However, it did consider that there was a need for improvement in the recruitment and training of D.R.O.'s.

I believe that a consideration of Australian conditions in the light of this report reveals a need for specially trained vocational counsellors serving within hospital rehabilitation units and autonomous rehabilitation centres, under the supervision of the medical leaders of such institutions. However, such counsellors must be able to undertake field work, and, of course, to act in liaison with, and complementarily to, placement officers (D.R.O.'s).

The Over-all Problem.

Surveys of the work of the D.R.O. service show that some 70% of clients are placed within six months, a figure which correlates well with certain findings of Ferguson and McPhail (1954). (See Appendix A.) These workers reviewed the fate of a large group of patients of all ages three months after they had left hospital, and found that one-third were still not back at work.

The problems of employment in rural districts and in the outlying suburbs are unique and not of ready solution. In many cases, so long as a patient lacks the financial resources to move his domicile, they are unable to be solved.

[1] Committee of Inquiry on the Rehabilitation of Disabled Persons, set up under the chairmanship of Lord Piercy in 1953.

There is a hard core of unemployable, mentally subnormal persons, and also a hard core of those who (often with some difficulty) can be diagnosed as having an inadequate personality. Suitable sheltered workshops are capable of absorbing into part-time remunerative work a significant percentage of the less unfit invalid pensioners, including even the mentally subnormal, the epileptic, and those with certain recurrent ailments (Editorial Article, 1958.) The percentages of people in such categories will vary from geographical area to geographical area, for many diverse reasons.

Sheltered Workshops.

Sheltered workshops cannot be superimposed upon a community, but must arise from within it. They usually need to be subsidized; but the subsidy should be so made as to encourage local community voluntary assistance.

Sheltered workshops need to be in the hands of competent, sympathetic but level-headed experts in this field of accounting. Suitable reliable and responsible trustees are likewise almost mandatory to assist in the safe direction of the affairs of a sheltered workshop. The actual operations of the latter usually rest most safely in the hands of an experienced foreman-manager.

Without suitable hostels, even the part-time employment of a sheltered workshop (not to mention the benefits of the *camaraderie* of fellow workers) is denied to very many of the disabled.

Finally, a sheltered workshop should ideally lie geographically near a suitable public hospital, so as to derive readily two desirable and almost essential benefits: on the one hand, reliable medical and almoner attention at short notice when necessary; and on the other, the services of competent honorary medical staff to assist the management in the wise selection of the "sheltered" workers.

These desiderata are not idle musings. They are the experiences of the sheltered workshops of South Africa (set up initially for ex-servicemen) (Department of Labour, 1954; "Life Begins at 70", 1953) and of the Abilities Building Centre, a sheltered workshop operating within the shadow of the Mayo Clinic.

Needless to say, pioneers have to combine many onerous functions and assume heavy personal responsibilities. This is not to say, however, that others less able and less energetic and less stable could be expected to "go and do likewise".

Epilogue.

The principle should be clearly seen that the problem of employment of the disabled is not a static one, and is not one susceptible of a centralized solution.

In theory—and it has proved so in practice—the basis of any solution must rest in hospital rehabilitation departments (and certain autonomous rehabilitation centres). It should be noted, however, that these will not be adequate until each is under full-time medical direction (Meyers, 1957), with adequate staff including vocational counsellors.

Such "peripherally" distributed working units can keep in touch with an ever-changing situation and, through the twin placement avenues of governmental placement officer and placement-vocational counsellor, can do the best most efficiently, most quickly and with least confusion.

Acknowledgements.

The opinions expressed in this paper are my own, and do not necessarily represent the views or policy of the Commonwealth Department of Social Services or of its Rehabilitation Service. This paper is published with the permission of the Director-General of the Department of Social Services.

Appendix A.

A recent valuable piece of research work has shown conclusively that in general the long-term outlook for post-hospital patients is not so good as is generally thought. Ferguson and MacPhail have made a valuable survey in Scotland of the post-hospital fate of a large group of patients. Five hundred and forty-eight men of all ages were interviewed in their own homes three months after leaving hospital; of these, 55·5% had returned to work, while 33·7% were still not yet back at work. The proportion back at work at the end of the three months varied with the nature of the work; among non-manual workers it was 74%, among skilled manual workers 58% and among unskilled manual workers 41%.

Duration of absence from work on account of the illness which had necessitated admission to hospital varied considerably with the nature of the illness. Among those who had been treated for alimentary disorders, 80% were off work for less than three months, whereas among those treated for diseases of the circulatory system, duration of absence from work was less than three months in only 33% of cases.

Of the men who were back at work, 21·5% were in jobs which could be described only as unsuitable, having regard to the demands of the occupation and the condition of the man. The proportion in unsuitable work was high among young men between the ages of 15 and 19 years and at ages over 50 years, when many suffered from heart disease.

Forty men in the series had been registered as disabled under the *Disabled Persons Act*, 37 of them prior to admission to hospital. Of those registered under the Act, only 32% were back at work three months after leaving hospital.

It can thus be seen that there is a need for a wider understanding of the problems and difficulties of post-hospital rehabilitation in all classes of case. Ferguson and MacPhail (1954) emphasize that the psycho-social are at least as important as the medical and surgical factors in these varied problems.

Appendix B: Explanatory Notes.

D.R.O.—In Britain the Ministry of Labour operates employment agencies, officers of which—the disablement resettlement officers—attempt to find suitable work for the disabled.

Resettlement Clinics.—At certain of the larger hospitals—and in particular at King's College Hospital, London—a meeting between doctor, sister, almoner, therapist (occupational therapist and/or physiotherapist) of the hospital and the D.R.O. is held—a resettlement clinic—to permit adequate exchange of information between those responsible for the rehabilitation of the patient and the D.R.O., and to help the latter in his efforts to find suitable work, not by any means for all patients, but for those whose placement, for medico-social and other reasons, presents difficulties.

TABLE I.

Great Britain.	Australia.
Ministry of Labour ..	Commonwealth Department of Labour and National Service.
Employment Exchange (of this Ministry).	District Employment Office (D.E.O.) of the Commonwealth Employment Service (C.E.S.) of this Department. There are offices in main suburban and rural areas.
D.R.O. Service ..	Physically (and Mentally) Handicapped Persons Section of the C.E.S. There is a headquarters office in Sydney, and in each D.E.O. there is an officer detailed to attend to the placement of the handicapped unemployed referred to, or required to attend at, the particular office.
D.R.O.	Placement Officer (for the disabled).

Vocational Counsellor.—Ideally, the vocational counsellor is a mature, stable adult, well versed in details concerning available educational facilities (agricultural, technical, clerical, primary, secondary and tertiary), and enlightened concerning the habits and practices of industry and commerce. Ideally, he should have had basic training comparable to that of an almoner, or be educated to, say, matriculation standard or its equivalent, with such basic training reinforced by certain special training. Ideally he should be expert enough in his own sphere to assist in the wise selection of the educational or vocational training of the trainable disabled, medically guided to ensure that the training and ultimate work will be within the patient's

physical and mental competence, and otherwise wholly practicable. Ideally, he should have an up-to-date and exact practical working knowledge of many aspects of legislation of the land and of institutions—for example, requirements for entry into training courses, correspondence schooling, union (and especially apprenticeship and slow workers') regulations, examination requirements, etc. Ideally, he should also be competent and able to maintain an up-to-date and wide first-hand knowledge of office and factory conditions. Ideally, through his personal characteristics he should be able to develop and maintain friendly relationships, not only with those with whom he must work within the rehabilitation unit, but also with principals of training colleges, personnel officers of industry and union officials.

Table of Comparisons.—The organization in Great Britain and that in Australia are compared in Table I.

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SOME PRACTICAL ASPECTS OF DEAFNESS.[2]

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CONSIDERATION of the hearing apparatus reveals quite clearly where and how various interferences with the hearing process may produce deafness. I shall not discuss such matters as obstructions in the external auditory canal, and, further, there seems to be little need to go into the obvious processes which may occur in the middle ear in catarrhal and suppurative inflammation, and there interfere with the normal transmission of sound vibrations across the drum. All will know of the need to overcome infective processes in the nose and throat, as well as in the Eustachian tubes, middle ear and mastoid cells, in an endeavour to bring to a halt the inflammatory reactions which interrupt or dampen the sound waves on their way to the inner ear.

Perhaps not all are yet aware that today much can be done to repair the ravages of inflammatory processes. In common with all the regions of the body, surgical endeavour in the ear has been given fresh encouragement under the protection of antibiotics. In aural surgery, the development of methods providing brilliant illumination and high magnification have led to scientifically conceived and precise methods of operating on these minute and delicate parts. Today we can safely clean out little pockets of disease around the oval and round window regions, aided by greatly enlarged vision. Should it be possible to eradicate infected foci in all these parts, it is with good expectancy of success that we may attempt to reconstruct a closed tympanic cavity or drum, with an outer vibratile membrane linked through a functioning ossicular chain to the oval window. While it is inappropriate to attempt to describe all the surgical procedures devised for this purpose, nevertheless, it is usefully illustrative, for instance, to explain that, when there is a persisting tympanic perforation, we are able to raise the outer epithelium around the margins of the perforation, thus rendering raw this area. A split skin graft taken from the patient's mastoid surface may now be cut to size and placed accurately over the perforation and its periphery that has been made raw, where in many instances it will adhere and grow, thus restoring nature's original tympanic structure. In a patient with both ears affected, to achieve this result in one or both ears may restore much of the lost hearing. In other instances a pedicled graft may be raised off the wall of the auditory canal.

The next form of deafness that I will again pass with but brief reference is "otosclerosis". This is a peculiar condition in which the delicate annular ligament about the footplate of the stapes becomes ossified, so that sound vibrations crossing the tympanum are damped, with the result that insufficient force is passed to the cochlea, where the vibratile motions are converted to nerve impulses. The obvious answers to this are (i) to put the vibrations in with greater force (by a hearing aid), (ii) to make a new window close to the obstructed one (fenestration), or (iii) to try to render mobile again the fixed stapes footplate. It is common knowledge that a good hearing aid can give great assistance to a deafened person. Unfortunately, a hearing aid does not cure or even retard progressive deterioration of hearing. It is for this reason especially that surgery remains a worthwhile endeavour, for although the results of operation are not always fully equal to normal, nevertheless successful operations do seem in very many cases to produce a lasting benefit. Success in human endeavour is not always complete. Thus it is that efforts continue with the object of simplifying the procedures whilst taking minimal risks of harm-doing in the process and, of course, always seeking a higher percentage of success. A practical operation for stapes mobilization has always been the hope of the aural surgeon. For otosclerosis, the commonest form of bilateral deafness, some means to free the fixed stapes is the obviously desirable objective. Today that objective can be achieved in a good number of cases. This operation is definitely worthy of trial in every case in which there is a reasonably functioning inner ear and eighth nerve. Essentially, the objective is to try to fracture the somewhat brittle otosclerotic bone that has formed in the annular ligament, which suspends the stapes footplate in the oval window. Access to the middle ear is gained via a rather cramped approach along the posterior wall of the external auditory canal, the tympanic membrane being then detached. In the simplest cases pressure is applied to the neck of the stapes with perhaps a little rocking movement; with a little cracking sound the stapes may be caused to move, and can be rocked minutely in its bed. It is not always as easy as this, for sometimes, rather than crack free in the oval window, the stapes itself will fracture, and although fresh mobility is to be seen, this is not imparted to the oval window at all and hearing is no better. Attempts are now made under high magnification to lever the stapes footplate free, using minute picks and levers. Obviously, when these extremely delicate movements have to be tried, bone trimming is often necessary to gain good visibility, and the operation is robbed of simplicity. A successful mobilization operation has this advantage—the period of stay in hospital is short (two or three days), incapacity is minimal, and a dry ear results. Should the functional gain be disappointing, a fenestration can still be performed without any lessening of the chances of success, or a hearing aid may still be usefully employed. In other words, the operation does little or no harm. In the doing, stapes mobilization is a delicate process requiring much practice for the acquisition of skill and confidence. In selected suitable cases not many more than 50% of the operations prove successful. Nevertheless, when a surgeon has made himself able to recommend the operation, confident that he can perform it without doing harm, success in half his cases is reasonably encouraging.

Another group of cases requiring discussion comprises cases of deafness due to disturbances of the inner ear and the eighth nerve—what we call nerve deafness. It is important that general practitioners should understand these conditions, for it is to them that many will go for

[1] Journal of the British Empire Service League, South Africa.

[2] Read at a post-graduate course in Sydney on July 18, 1959.

advice in the first place; yet it is likely that it will be only in the early stages that it may be possible to do something to save the victim's hearing. The minute functioning elements of the inner ear are strangely interesting in that individual sensitivity is a proven fact, especially in the matter of damage from noise or vibration. It is extraordinary, too, that there are toxic substances which are selective of the cells of the inner ear.

Amongst a group of workers in a noisy machinery shop, most seem able to face the same noise environment day after day without harmful effects. In the armed services we may find artillerymen, machine gunners and tank and aeroplane crewmen who suffer little or no ill effect from noise and blast; yet there are individuals who suffer lasting damage from a day at the rifle range, and who will inevitably become more deafened with continued exposure. How are we to detect these vulnerable people? In the industrial world it is a foolish employer, owning any kind of noisy works, who fails to have all employees tested on their engagement, and then to have further screenings to detect those who may be commencing to be affected. It is an interesting fact that deafness due to noise commonly shows its earliest and greatest effects about the 4000 cycle tone pitch, so that subsequent screenings may need periodical retesting only at this frequency. Where does the general practitioner come into this picture? Not all workers are occupied in big factories where screenings may be a routine procedure; indeed, the potential victim may be self-employed in his own garage or workshop. He may be a farmer who uses his own motor-tractor, harvester or chaff-cutter, or he may go in for rifle or gun shooting, or perhaps he is a young man who likes to tune his motor-car or bicycle. Such individuals may come to the general practitioner complaining of tinnitus and slight deafness. He may test the blood pressure, and perhaps exclude the general run of progressive constitutional diseases. It is not unreasonable as well that he should have a watch of known hearing distances, and match the patient's ears separately against the watch. If the hearing is down, even a little, the relative hearing at the ear and on the mastoid bone may be tried with the 512 or 1024 tuning fork. Some measure of deafness may well be detected. When this is done, by the general practitioner, careful inquiry and a timely warning against the damaging effect of rifle fire, machinery, etc., may help to save that man's hearing. A farmer may be warned always to have a good thick rubber cushion on the seat of his tractor, to wear protective ear plugs when common sense suggests it, and to avoid noise and vibration whenever possible. Similar advice may save the hearing of many others exposed to the risks of damage.

The peculiar fact of individual susceptibility to noise and blast damage is striking, and stimulates the desire to know why.

What is perhaps more intriguing is the fact that there are potent biochemical toxins which particularly select the inner ear tissues for their damaging effect. Amongst infective diseases, mumps and measles are prone to damage the hearing severely and rapidly. Quinine, salicylates and some of the complex arsenicals, such as arsenobenzole, have long been known as potential threats to the ears. It is when we come to the antibiotics that the most striking examples of all are encountered. It was early found that prolonged treatment with streptomycin was prone to affect the balancing function, and that it would, if persevered with, soon cause loss of function in the semicircular canals. In the search for a modification which did not carry this threat, dihydrostreptomycin was evolved. How strange it was that this substance particularly selected the hearing, and if persevered with caused severe irreversible deafness! Neomycin is another antibiotic possibly more damaging to the hearing than any. This substance is but rarely used internally, and when it is used as a topical application in ointments, etc., the danger is not great.

The strange fact of selectivity is the interesting feature. The antibiotics act against bacteria by interfering with their intracellular metabolism. It is thought that they may paralyse certain intracellular enzymes. Now it is believed that the vitamins act in some way as enzymes, and vitamin B complex is thought especially to be of particular importance to specialized nerve tissues. Japanese investigators in particular have reported that streptomycin can be administered without harmful effects for quite long periods if large doses of vitamin B are given throughout the treatment. While there is no certainty of the mode of action of these toxins, and no guarantee that vitamins will always be the effective antidote, yet it seems worth while, when such a dire consequence as severe deafness is possible, that we should endeavour to protect all patients with mumps or measles, and all exposed to streptomycin or neomycin, by giving them large doses of vitamin B complex parenterally, then orally, during and for some time after the period of exposure to risk. Let it be remembered that the damaging effect may continue to operate for some time after the toxic substances have been withdrawn. Should symptoms of inner-ear disturbance develop in either the toxic or the noise-trauma cases, it is as well, too, to administer capillary vasodilators, such as nicotinic acid (50 to 100 mg.), by mouth, on an empty stomach, every six hours, or by intramuscular injection. The hot, tingling flush aimed at is but momentary, and appears to be harmless, yet may contribute to the saving of hearing function.

Deafness, with and without vertigo, but often with disturbing tinnitus, may, of course, be associated with hypertension, and perhaps with localized vascular insufficiency. The symptoms may respond somewhat to successful hypotensive measures. Tinnitus is often an obstinate though variable symptom, and the deafness may progress insidiously, or at times in sudden episodes of further loss of function. The administration of the B group of vitamins and tranquillizing drugs and general hypotensive measures may wage only a delaying action against the effects of vascular disease, as in other parts of the body.

I shall now briefly mention lesions of the eighth nerve itself. Traumatic damage from skull fractures and penetrating wounds will be obvious, and is irreversible. Tumours of the eighth nerve are amongst the commonest intracranial new growths. The lesion causes disturbances of function, such as tinnitus, vertigo and deafness, but may come on very slowly. The effects usually are unilateral. Typically the hearing loss is of the perceptive type, with reduced bone-conduction hearing. The vestibular function is greatly reduced, or lost altogether, on the affected side. Certain closely placed nerves are likely to be involved early. Facial weakness, strangely enough, is not an early sign. Pressure on the fifth nerve may quite early cause homolateral facial tingling, numbness and loss of sensation, while the corneal blinking reflex may be dulled.

Most of these patients will be referred to the otologist and thence to the neurosurgeon for final diagnosis and operation. All practitioners, however, should be ready to suspect such a lesion in the presence of unilateral deafness and tinnitus. A brief check over the responses from adjacent nerves may add strength to suspicion. These tumours, if found early, can be successfully operated upon. Like all growths, too late recognition assures disaster to the unfortunate victim.

Conclusion.

I have endeavoured to touch briefly upon some of the recent advances in the management of deafness, so that the general practitioner may be best fitted as the family adviser to guide the patient to the best methods of protection against hearing loss and treatment when necessary. I think that in such diseases as mumps and measles and when there is exposure to noise, the general practitioner should take definite steps towards prevention of aural damage in the manner outlined. If he is able early to recognize a possible eighth nerve tumour, he may save a patient's life, and perhaps his hearing. One hopes that he will find something of use in this all-too-brief summary of some of the problems of deafness.

Reviews.

A Manual of Psychiatry. By K. R. Stallworthy, M.B., Ch.B.; Fourth Edition; 1959. Christchurch: N. M. Peryer Limited. 7¼" x 4¾", pp. 365. Price: 30s. (New Zealand).

IN his "Manual of Psychiatry", Dr. K. R. Stallworthy presents his subject clearly and concisely, and is as up-to-date as can be expected with the steady appearance of new tranquillizers. Does the fact that one of similar twins stays healthy prove that a favourable environment may "avert illness even in the strongly disposed"? Puerperal obsessional states are deemed legitimate indications for abortion. Confusional states which complicate an established dementia are unfortunately "heralds of death" in the experience of the institutional psychiatrist. The causes of these senile confusions are discussed, and if they are recognized early the prognosis is far more favourable. Apart from indications, the leucotomies are dismissed with the words "certain parts of the brain are cut". A chapter is devoted to nursing procedures, pre-operative and post-operative care and other duties of nurses in psychiatric hospitals. There is a chapter on the fairly simple legal provisions in New Zealand; testamentary capacity is discussed, but not criminal responsibility. Psychotherapy is covered in the final chapter of 12 pages.

The appearance of a fourth edition shows that this work fills a need. It is an accepted textbook for mental nurses in New South Wales and elsewhere, and medical students will find it informative and reliable.

Protides of the Biological Fluids: Proceedings of the Sixth Colloquium, Bruges, 1958. Edited by H. Peeters; 1959. Amsterdam, London, New York and Princeton: Elsevier Publishing Company. London: D. van Nostrand Company, Limited. 9¼" x 6¾", pp. 342, with many illustrations. Price: 45s. (English).

A COLLOQUIUM on various aspects of protein chemistry of biological fluids is held annually in Bruges, and the proceedings of the sixth colloquium held in 1958 have just been published. There is no indication anywhere in the book as to who organizes the meetings and what people attend; but the names of the members of the Scientific Committee are given, and it is evidently international. In the publication there are 51 papers and two round-table discussions. Sixteen of the papers are in German, 19 in French and the rest in English. Each paper has a brief summary in English. The field covered is very wide, but a great deal of space is taken up with descriptions of various methods of electrophoresis and chromatography and the results obtained by the use of these methods on various biological fluids. It is impossible in a short review to give an account of the contents of the 51 papers. There is very little of clinical interest in the papers, for most of them deal with science. The round-table discussions on "protein pattern in normal and pathological cases" and "electrophoretic techniques" are scrappy and not very good.

The book as a whole contains much that is of interest to the scientific worker on the proteins of body fluids, but little for the practising physician.

Gynecologic Endocrinology. By G. M. Riley, Ph.D.; 1959. New York: A Hoeber-Harper Book. 10¼" x 6", pp. 350, with illustrations. Price: $8.50.

THIS book is written for the general reader, and aims to help those involved in solving the everyday problems of gynæcological endocrinology. In general, Dr. Riley achieves a disarming simplicity of style, and the presentation of the facts is such that it is easy to find one's way around the book. The author begins with a rather too brief account of endocrine physiology, and then describes the major diseases with the aid of numerous lists, tables and clear illustrations. The method of writing a rather thin text around a number of tables and lists is now quite familiar and enjoys certain advantages. However, one of the drawbacks of this technique lies in the inevitable failure to highlight the cardinal differences between several diseases—differences which can easily be lost in a general list of signs, symptoms and laboratory data.

Two of the best features of the book are to be found near the end—namely, a splendid account of the important laboratory methods in current use, and a full list of the hormone preparations at present available. The laboratory methods are particularly well presented, and the approach to such complex procedures as œstrogen assay is realistic, in that the author shows something of the labour involved and is consistent in the intelligent and sparing use of these assays recommended in the text.

In the matter of treatment Dr. Riley is to be complimented. His statements are eminently sane, avoiding the extremes of fussy over-treatment on the one hand and nihilism on the other. It is refreshing to find an author who does not regard dilatation and curettage as a cure for every menstrual disorder.

A chapter on infertility presents a useful outline of the general plan of attack used in investigating and treating this group of disorders. Another chapter deals with general endocrine syndromes: this is very sketchy, and seems out of place in such a book.

One striking feature of this book is the lack of emphasis upon psychological factors in gynæcological endocrinology. Perhaps this is partly related to the method of presentation mentioned above. It may be that some authors have gone too far in emphasizing the psychopathology of endocrine disorders; but those who deal with patients suffering from these disorders can hardly fail to realize the paramount importance of psychological factors in the ætiology and in the response to treatment of gynæcological endocrinopathies.

The Meaning of Poison. By Lloyd G. Stevenson, M.D.; 1959. Lawrence: University of Kansas Press. 8¼" x 5¼", pp. 64. Price: $2.00.

IN these two lectures, Lloyd G. Stevenson, Professor of the History of Medicine and Dean of the Faculty of Medicine at McGill University, Montreal, discusses his subject with considerable erudition. He points out that the lectures deal with two aspects of the meaning of "poison"—poison as a pathogen in a very broad sense, which he believes to be the most important of the connotations of the word, and poison as a tool of research in physiology and pharmacology. In the first lecture, under the title "Poison, Infection, and Contagion", he seeks out the closely related ideas lying behind these three words, or their equivalents, as they were known in the ancient world, and traces them through to near modern times. The title of the second lecture, "Hellish Oorali", comes from what Stevenson charitably describes as "a rather bad poem" by Alfred Lord Tennyson entitled "In the Children's Hospital". "Hellish Oorali" is curare. Again quoting a variety of unusual sources, Stevenson sets out the history of this potent drug from the jungles of South America through to the early European experimental work in the eighteenth century and the investigations of Claude Bernard in the mid-nineteenth century, and finally to its modern use as a relaxing agent in anæsthesia. In both lectures Stevenson slips into unfrequented by-ways and produces many little-known facts. The lectures are stimulating as well as informative.

A Synopsis of Skin Diseases. By Bethel Solomons, jr., M.A., M.D., F.R.C.P.I.; 1959. Bristol: John Wright & Sons, Limited. 7¼" x 4¾", pp. 304, with 14 illustrations. Price: 30s. (English).

THIS book, as is pointed out in the preface, is a companion to the others in the "Synopsis" series. The book is intended for the busy practitioner, for those who have to revise the subject rapidly and also for the student about to take his final examination. It does not replace the larger scope of a textbook. In our opinion, it fulfils its aims very satisfactorily. As the author has pointed out, it is impossible in these days to keep pace with all the advances both in the understanding and in the treatment of disease; but this book is remarkably up to date, and also is very complete in the diseases of which it treats. The subject matter is very well set out, divided by clear headings and easily readable. Naturally, in a book of this nature illustrations are not to be expected; those that are present deal mainly with the diagrammatic representation of regional sites in which diseases commonly occur, and this feature should be of great help to the student.

Australian dermatologists would not agree with all the details of the treatment recommended in every instance; for example, the strength of local applications used is much higher in the colder climate of the United Kingdom than can be recommended with safety in this temperate and subtropical zone. Also the use of undecylenic acid ointment in the treatment of tinea cruris is not very well tolerated by patients in this part of the world. One would certainly not agree with all the observations of the author in regard to radiotherapy, and very few dermatologists in this country would agree with his statement that cautery in the treatment of squamous celled carcinoma of the skin is "effective in skilled hands". We believe that the treatment is either surgical or by radiotherapy.

The effects of sunlight in the production of melanomata of the skin is not mentioned. In the rather complete list of skin manifestations accompanying internal cancer, exfoliative dermatitis in association with it does not find a place. Though this has been reported in the Australian dermatological literature quite recently, most authorities do not now use arsenicals in the treatment of syphilis, being quite satisfied to rely upon antibiotics for extermination of this disease. One other criticism in therapy that could be made is the omission of smallpox vaccination repeated at two-monthly intervals in the treatment of herpes simplex recurrens, which has been found quite an effective means of treatment.

This book certainly has a place in the reference library of a busy practitioner, and would be found, we believe, more useful than a more expensive atlas of skin diseases. It is recommended.

Handbook of Diet Therapy. By Dorothea Turner; Third Edition; 1959. Chicago: The University of Chicago Press. 9" x 6", pp. 237. Price: $5.00.

THERE are many books on diet therapy, some good, many very poor; but a book which goes into three editions in twelve years must have some appeal. This is the case with the present book. The qualifications of the author assure one that the data presented are as accurate as can be in the present stage of knowledge. She is Chief Medical Nutritionist in the Department of Medicine, University of Chicago, and editor of *The Journal of the American Dietetic Association*, and she was formerly chairman of the Diet Therapy Section of the American Dietetic Association. The method of treatment of the data is somewhat unusual, but very rational. The diets for all types of conditions are built around what may be called optimal normal nutrition. Foods have been classified into four main groups, and in making up a diet something must be taken from each group. A fifth group—fats and sweets—has been considered separately, mainly for making up calories. The four groups are as follows: milk group; meat group; vegetable and fruit group; bread-cereal-potato-legume group. These supply the main part of the recommended protein, mineral and vitamin intakes for the healthy individual.

In making up different dietary patterns, care is taken to ensure that they are at least as good as an adequate normal diet in proteins, minerals and vitamins. The normal diet having been discussed on such a plan, the modifications of the basic diet necessary at various phases of life are stated. Numerous tables are given, in which every aspect of foodstuffs usual in the American dietary is detailed. Then follow discussions on various modifications of the normal diet for special conditions. These include modifications in consistency, in calories and in fat intake, protein modifications such as phenylalanine restriction and gliadin restriction and modifications in protein-fat-carbohydrate content, as well as the sodium-restricted diet, the low-purine diet and the ketogenic diet. It will be seen that little is left out of consideration. After some study to become used to the method of formulation, it should be possible, without much trouble, to make up varied dietaries for any condition in which special diets are considered necessary. There is a useful section on interviewing the patient, and there are more than 50 tables of various types of data.

This book is thoroughly practical if one will spend a little time in understanding its methods; it will be of great value to dietitians in particular, but also to physicians concerned with the dietary management of patients.

Squint and Allied Conditions. By George P. Guibor, M.D., D.D.S.; 1959. New York and London: Grune & Stratton, Inc. 8" x 5¾", pp. 368, with 99 illustrations. Price: $11.50.

A PRACTICAL man of long experience always has something worthwhile to pass on. The virtues to be found in this book spring from sound, sympathetic, practical experience, so that there are many points of stimulating interest and usefulness in the author's approach to both the diagnosis and the treatment of squint.

The weaknesses of the book are, to some extent, anticipated in the preface. The theoretical approach is doubtful, and the audience is a mixed one of oculists, orthoptists and opticians; this leads to the inclusion of material that is unsuitable for one group and irrelevant for another.

There is great emphasis on the use of prisms, not only for diagnosis and measurement, but also in the treatment of convergent squint, and the surgical bias is towards shortening operations on inefficient muscles rather than towards weakening of their opponents.

There is very little on the handling of squint in the vital early months of infancy; the tendency is to treat squint rather as a well-established phenomenon. This, of course, is what it becomes only too readily when such an approach is made.

The book is a handy size and well and clearly set out, and it contains only a few misprints. As a treatise on the practical handling of established squint it is valuable, honest, helpful and useful; but it is not a textbook on squint.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Process of Aging in the Nervous System", by Thirty-Two Contributors; edited by James E. Birren, Ph.D., Henry A. Imus, Ph.D., and William F. Windle, Ph.D., Sc.D.; with a foreword by Pearce Bailey, Ph.D., M.D.; 1959. Oxford: Blackwell Scientific Publications. 9" x 6", pp. 238, with 42 illustrations. Price: 52s. 6d. (English).

"Methods of Radiochemical Analysis", Report of a Joint WHO/FAO Expert Committee; World Health Organization Technical Report Series, No. 173; 1959. Geneva: World Health Organization. 9" x 6", pp. 116, with illustrations. Price: 5s. (English).

"Experimental Surgery: Including Surgical Physiology", by J. Markowitz, M.B.E., M.B. (Tor.), Ph.D., M.S. in Exp. Surg. (Minn.), J. Archibald, D.V.M., M.V.Sc., Dr. Med. Vet. (Giessen), M.R.C.V.S., and H. G. Downie, D.V.M., M.S. (Cornell), M.V.Sc.; fourth edition; 1959. Baltimore: The Williams & Wilkins Company. Sydney: Angus & Robertson, Limited. 9" x 5¾", pp. 944, with 580 illustrations. Price: £6 17s. 6d.

"The Surgical Treatment of Facial Injuries", by Varaztad Hovhannes Kazanjian, C.M.G., D.M.D., M.D., F.A.C.S., D.Sc. (hon.), and John Marquis Converse, M.D., F.A.C.S.; second edition; 1959. Baltimore: The Williams & Wilkins Company. Sydney: Angus & Robertson, Limited. 10" x 6½", pp. 1124, with 1155 illustrations. Price: £12 2s.

"Clinical Orthopædics, 13", Anthony F. DePalma, editor-in-chief, with the assistance of the associate editors, the Board of Advisory Editors and the Board of Corresponding Editors. Number Thirteen, Spring, 1959. Philadelphia and Montreal: J. B. Lippincott Company, Limited. Sydney: Angus & Robertson, Limited. 10" x 6¾", pp. 406, with many illustrations. Price: £3 6s.

"First International Congress of Neurological Sciences: Brussels, 21-28 July, 1957", edited by L. van Bogaert and J. Radermecker; Volume 3; 1959. London, New York, Paris, Los Angeles: Pergamon Press. 10½" x 7½", pp. 707, with illustrations. Price: £7.

"Maudsley Personality Inventory (Test Set)", by Professor H. J. Eysenck; 1959. Melbourne: Hodder & Stoughton, Ltd. Price (Australian): Test 8d. per copy, 6s. 6d. per dozen; Key E Scale, Key N Scale, 1s. 11d. each; Manual 4s. 9d.

"A Treatise on Hygiene and Public Health: With Special Reference to the Tropics", by Birendra Nath Ghosh, F.R.F.P. & S. (Glas.), F.R.S. (Edin.); edited by Rabindra Nath Gosh, M.B., B.S., D.T.M. (Cal.), D.P.H. (Lond.),; fourteenth edition; 1959. Calcutta: Scientific Publishing Company. 8½" x 5", pp. 955, with 163 illustrations and many tables. Price: 40s.

"Bonding of Fractures by Plastic Adhesives: With Further Applications in a Research Project", by Bernard Bloch, M.B., Ch.B. (Rand.), F.R.C.S. (Eng.); 1959. Sydney: Distributed by Surgical Research Centre, Prince of Wales Division of Sydney Hospital. 9½" x 7", pp. 24, with 24 illustrations. Price: not stated.

"Antibiotic and Sulphonamide Treatment: A Short Guide for Practitioners", by A. R. Anscombe, M.S., F.R.C.S., D. B. Brown, M.B., M.M.S.A., M.R.C.O.G., D. M. Davies, M.R.C.P.; edited by M. E. Florey, M.D., with a foreword by The Rt. Hon. The Lord Evans, G.C.V.O., M.D., F.R.C.P.; 1959. London, New York, Toronto and Melbourne: Oxford University Press. 6½" x 4", pp. 160. Price: 17s. 6d. (Australian).

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POPULATION PRESSURE.

IN the 1958 Rede lecture, Sir Charles Darwin, F.R.S., made this statement: "The present increase in world population holds threats for the future, outweighing completely any of the menaces that preoccupy statesmen and politicians." Everyone is aware of the menace of nuclear explosions, and attempts are being made to prevent their use. Although there are a few notable exceptions, such as Sir Charles Darwin himself, Sir Macfarlane Burnet and Aldous Huxley, few seem to be aware of the danger of overpopulation, and practically no attempts are being made to combat this menace. The subject is shelved by governments, although India and China do realize that overpopulation is cutting across their plans for development. The churches have shown interest in the problem but have not yet put forward a solution. The problem is rarely mentioned in universities, and is largely ignored by the great research foundations. The attitude of science in general appears to be that it is not at present important in the countries where science flourishes, although on the world scale overpopulation is probably the greatest biological problem of our time. Not only does it bear misery in its train in the overpopulated countries, but the rest of the world must eventually be involved in the repercussions. On a small scale, we can already see the repercussions—for example, the Notting Hill riots in London are the direct result of the overpopulation of certain West Indian islands.

Those who rightly distrust categorical statements will want to know why overpopulation should be called the greatest biological problem of our time. There is a biological law, which is no more than common sense, that the numbers of any animal must keep in balance with its food supply. If for any reason numbers outrun the food supply, nature will inevitably correct the imbalance by reducing the numbers. In the Stone Age, the human population with its hunting economy was small. Food was difficult to obtain in such a spear-to-mouth existence, but numbers were kept in check by famine, by war and by pestilence. With the advent of an agricultural economy, food was more easily obtained, and the human population slowly increased from about 200,000,000 in A.D. 1 to about 350,000,000 in A.D. 1600. From then onwards numbers increased more rapidly. In A.D. 1650 the rate of increase was such that the population would have doubled in about 200 years, but today, if present trends continue, the world population will double in 50 years, resulting in a population of 5,000,000,000 by the end of this century. Further, the rate of increase is still accelerating, so that estimates of world population in the future are continually being revised upwards. Today 40,000,000 people are being added to world population every year, and there is no evidence that deceleration of the rate of increase is yet in sight.

There are a number of reasons for such a tremendous increase in population in the last fifty years, but there is little doubt that the main reason is the conquest of the infectious diseases. These diseases, which in the days before the Pasteurian revolution took such a heavy toll of infants and children, can now be largely prevented. The greatest concentration of population is in Asia, and until recently Asian countries had a high birth rate and a high death rate. The introduction of preventive measures to control the infectious diseases resulted in a dramatic fall in the death rate; and as the birth rate remained high, the population is increasing by leaps and bounds. And preventive medicine has still some distance to go in these countries. For example, in 1950-1952 the expectation of life in India was 32·5 years, while in England it was 66·5 years. We must, of course, encourage such a development, even though these Asian countries are finding it more and more difficult to feed their rapidly increasing populations. In Europe, North America and Australia the death rate fell in this period, but there was a great decline in the birth rate beginning about A.D. 1880. The parallel fall in both death rate and birth rate resulted in a very slow increase in population in these countries; in fact, until quite recently the population of France was actually decreasing.

Turning now to the food supply, we can probably agree that in many lands there has always been a shortage of food despite the fact that the production of food has increased through the centuries. Today FAO (the Food and Agriculture Organization of the United Nations) estimates that two-thirds of the world's population is existing on less than 2200 Calories a day, and that the deficit of food is increasing. This may seem incredible to us, but although we are well fed, over half of the world's population lives in Asia, and over 70% of the underfed people are concentrated in that continent. A further 18% of the underfed live in Africa and Latin America. The cry "Are we our brothers' keepers?" has echoed down the ages. However sympathetic we may feel, the very size of the problem is so staggering that no easy solution seems possible. There is a surplus of food in some of the more fortunate countries, but even if there was a free distribution of these surpluses, it would hardly begin to feed these multitudes.

It has been calculated that if all the arable land in the world was cultivated to its maximum potential, there would be enough food for a world population of 7,000,000,000 people, if the food could be distributed equably and the question of payment was ignored. The "ifs" in this calculation cannot be passed over lightly. There is no doubt that the underdeveloped countries could improve their crop yields if the necessary capital, education and skill were available. FAO is working along these lines, but it will take time even under the most favourable conditions, and meanwhile the population is increasing much faster than extra food is being produced. It is much easier and quicker to cut down the death

rate by improved sanitation, hygiene, immunization and drug control than it is to increase the supply of food by better agriculture.

There is a natural tendency for overpopulated countries to look with longing eyes at the so-called "empty" lands, like Brazil or Australia, as a home for their surplus people. Apart from the enormous difficulty of settling immigrants into a new country such as Northern Australia (which is hardly a land flowing with milk and honey), the maximum immigration into Australia over a period of some years could not exceed 15,000,000. Compare this figure with the annual increase of some 25,000,000 in Asia, and one can see that mass immigration is no solution.

This then is the situation. First, in those countries mainly in Asia which contain more than half the world population the birth rate remains high, and the death rate is steadily falling; this results in a rapidly increasing population in countries which in the main are already overpopulated. Second, the food supply in these countries is inadequate now, and the imbalance between population and food supply is worsening every year because population is increasing faster than extra food is being and can be produced. Third, mass immigration from overpopulated countries into less populated lands is not a practical solution, because immigration could not possibly cope with the 40,000,000 annual increase of population.

What other solution is there for what has been called here the greatest biological problem of our time? The problem has been solved in those countries which, by intensive efforts, have evolved a civilization in the last two centuries which we call Western civilization—Europe, North America, Australia. Here the birth rate since A.D. 1880 has steadily fallen contemporaneously with the fall in the death rate, resulting in a fairly static population. Indeed the rise in the standard of living in a country like England would not have been possible if the population had continued to increase as it did in the first three-quarters of the nineteenth century. Why has the birth rate fallen since about 1880? This is not entirely clear, but birth control by contraception was without doubt a major factor. Is birth control the solution for the countries where the population-food supply balance has got out of hand? In theory it would seem so to many people; to others it is unacceptable. Moreover, one should not minimize the difficulties of introducing birth control on a large enough scale among people who on the whole are not well educated and are steeped in tradition, family customs and religions which are inimical to such a practice. Further, we must concede that a suitable contraceptive for these countries is not available. Some work is being done to find and test an oral contraceptive, but so far without much success. It is not a popular subject for research as is obvious from a perusal of projects supported by research funds. There are of course fashions in medical research, as in all human activities.

There is such a veritable chorus of silence on this subject that one naturally wonders whether the importance of population pressure is being exaggerated by those who have studied it. Or have we a situation parallel to the attitude of England during the arming of Hitler's Germany—a refusal to face the facts? If present trends continue, not only increasing undernourishment but starvation seems eventually inevitable in the threatened countries. Again pursuing the parallel of England in 1940, we may well ask if there must be actual starvation before the situation is appreciated and resolutely faced.

Current Comment.

EPIDEMIC EXANTHEM AND POLYARTHRITIS.

SINCE J. R. Nimmo[1] observed "an unusual epidemic" at Narrandera in 1928, information on "epidemic polyarthritis", as P. G. Dowling[2] named it in 1946, has slowly accumulated, and in this issue R. E. Shope and S. G. Anderson (see page 156) bring forward serological evidence that the responsible agent is a virus belonging to group A of the arthropod-borne, or arbor, viruses. This is an important finding and will encourage investigators in their endeavours to isolate the actual virus. Two arbor viruses of group B are known to invade Australia from time to time—those of dengue and Murray Valley encephalitis—but this is the first evidence here of a virus belonging to group A.

The disease has a wide distribution. Shope and Anderson list localities from New Guinea to the Murray, and the list could be extended. B. Short,[3] who was closely associated with Northern Territory cases, recognized five cases in Emerald, central Queensland, in April and May, 1949, and others during the Bougainville campaign. J. R. Nimmo[4] saw cases in the Torres Straits. D. J. R. Snow[5] reported that outbreaks had occurred in several country towns in Western Australia in February, 1956, as well as a few cases in Perth.

How is it spread? Mosquito transmission has been a favourite hypothesis from the start and is rendered still more likely by the recognition of the agent as an arbor virus. E. L. Morgan,[6] while collecting further information about Nimmo's epidemic, listed the mosquitoes that had been found in Narrandera. W. D. Pryor[3] identified mosquitoes collected during Dowling's epidemic. E. J. Britten[7] has published a valuable survey of mosquitoes prevalent in Western Australia. These and the many other locality records give useful leads in the search for the vector species.

E. W. Sibree's description of the disease on the Atherton Tableland led C. H. Knott[8] to recall a similar epidemic in that area in about 1940. It is tempting also to make a retrospective diagnosis of polyarthritis for cases (unpublished) seen frequently by Brisbane practitioners in the early months of 1940. They were characterized by a mild fever, rash and joint pains and were commonly, but with many reservations, labelled rubella. Might we have in polyarthritis an explanation for some "second" attacks of measles or rubella?

Perhaps the history of this disease goes back as far as 1886. In July of that year, Weber[9] reported an outbreak of "dengue fever" at Natimuk in western Victoria. He had seen 32 cases in the previous four months, which, allowing for publication time, implies summer and autumn. A diagnostic battle developed. Some patients

[1] MED. J. AUST., 1928, 1: 549 (May 5).
[2] MED. J. AUST., 1946, 1: 245 (February 23).
[3] MED. J. AUST., 1949, 2: 762 (November 19).
[4] MED. J. AUST., 1946, 1: 380 (March 16).
[5] Report of the Commissioner of Public Health, W.A. for 1956, 1958: 63.
[6] Report of the Director-General of Public Health, N.S.W. for 1929, 1930: 113.
[7] Report of the Commissioner of Public Health, W.A. for 1956, 1958: 64.
[8] MED. J. AUST., 1944, 2: 565 (November 25).
[9] Aust. med. J., 1886, 8: 291 (July, 15).

removed to hospital were declared there to be suffering from typhoid fever, but Weber presented the temperature records to the Central Board of Health, who pronounced them dengue. Weber examined blood and fæces bacteriologically—surely noteworthy enterprise for a country practitioner in 1886—but was unable to find a causative bacillus. It is true that Weber's description accords much better with dengue than with polyarthritis, though he may perhaps have based his description on the most severe cases. He noted, *inter alia*, a sudden onset, temperature rising to 103° F. or even 104° F. and pains in the eyeballs. Erythema and swelling of the joints were transitory. Epidemiologically, however, a diagnosis of dengue in Natimuk is unacceptable. No dengue epidemic has come nearer than Bourke, 500 miles away, and *Aëdes ægypti* has not been found south of the Murray. But some arbor virus may well have been responsible for the Natimuk outbreak. Shope and Anderson suggest that possibly more than one group A virus may be in Australia. This is plausible in view of the very wide geographical range and the variation in symptom-emphasis in different outbreaks.

THE RADIATION REACTION IN THE VAGINAL SMEAR.

THE important subject of radiation reaction in the vaginal smear has been discussed in a recent supplement to *Acta Radiologica* by Olle Kjellgren.[1] After surveying the literature on gynæcological exfoliative cytology from an historical point of view, he goes on to consider methods of obtaining specimens, fixation techniques and staining procedures before presenting the main subject matter.

The effect of radiation on the vaginal smear was first described by Ruth Graham in 1947, and Kjellgren describes her findings and those of others. He then presents the results of his studies in two series of cases, in both of which the patients received combined radium and deep X-ray therapy according to the Stockholm technique. Series 1 consisted of 241 patients from whom smears were taken prior to the second or third radium application. Series 2 consisted of 46 patients living near the clinic who had smears taken three times a week.

The estimation of the radiation response was based on the percentage of benign exfoliated epithelial cells which showed radiation effects. This response was considered good when 60% or more of the exfoliated cells exhibited any of the following changes: (*a*) Size increase: a major axis greater than 35μ for basal cells or 60μ for intermediate or superficial cells. (*b*) Nuclear changes: multinucleation, deformity and karyorrhexis or, in a basal cell, karyopyknosis. (*c*) Cytoplasmic changes: vacuolation, bizarre shapes and, in the case of basal cells, modified staining properties.

In Series 1, a 36·5% five-year cure rate was found in the group with poor radiation response as determined by the vaginal smear, and a 73·9% five-year cure rate in the group with good response. It appeared that the radiation response was unrelated to the staging of the tumour, the duration of history, the state of parity or the radium dosage within therapeutic limits. The rate of primary healing (no sign of tumour on clinical examination of the cervix) was even more significantly related to the cytological response. The local five-year healing rate was 91·3% in the good response group, against 50·8% in the poor response group. Among the patients who died during the five years of observation, those in the good response group tended to survive longer than those in the poor response group.

In Series 2, the changes in the radiation response were considered with respect to time. The maximum changes were present about the twelfth day after the first treatment and ten and a half days after subsequent treatments. The radiation reaction in the vaginal smears taken at the time of the first and second radium insertions was representative of the actual radiation reaction in most cases, although 14% of those whose serial smear showed a good response did not show a frequency equal to or exceeding 60% at the time of later radium treatment. So, in Series 1, some good response cases were probably missed.

With regard to hormonal factors, it was found from the basal cell index and the karyopyknotic index that the good response patients had lower initial œstrogen activity and that their œstrogen activity diminished more rapidly during the course of treatment. Patients with low hormonal activity showed a significantly lower poor response frequency than those with high activity. It is concluded that the cytological response is correlated with the prognosis, in the sense that a good response is associated with a higher local healing rate and hence with a higher five-year apparent recovery rate. It seemed in these studies to be uncorrelated with the incidence of metastatic spread. If a good cytological response fails to materialize under ordinarily favourable conditions, the prognosis will be poor. If a good cytological response is obtained in otherwise unfavourable circumstances, the prognosis will be good.

[1] "The Radiation Reaction in the Vaginal Smear and Its Prognostic Significance: *Studies on Radiologically Treated Cases of Cancer of the Uterine Cervix*", by Olle Kjellgren, *Acta Radiologica*, Supplement 168; 1958. Stockholm: Acta *Radiologica*. 9½" x 7", pp. 170, with 26 illustrations, 1 colour-plate and 31 tables. Price: Sw.Kr. 35.

RABIES.

ALTHOUGH the number of deaths from rabies is small in all but a few parts of the world, the disease continues to be present in most countries. According to information collected recently from 75 countries for a WHO expert committee, rabies is present in 52 countries and not present in 23 (including Australia). The only countries with any considerable number of deaths in 1958 were India (205 deaths reported from the Haffkine Institute, Bombay, and 250 from the Pasteur Institute of Southern India, Coonor) and the Philippines (217 deaths reported); in the same year there were 43 deaths in Mexico, 30 in Egypt and 12 in Turkey. Tens of thousands of people are given prophylactic treatment against rabies every year.

The reported sources of infection are interesting. In the United States of America six deaths from rabies occurred in 1958 and five in 1959 up to November 27. Of the 11 cases, five are attributed to dog bites, three to bites from bats, one to a bite from a fox, one to a bite from a skunk and one to an unknown source. While in the U.S.A. the animal mainly responsible for transmission is the dog, more and more evidence has been brought forward to show the importance of the bat in maintaining the infection in wild-life and causing infection in man; the wild animal most frequently found infected is the skunk. In Germany, investigations tend to show that the bat is not important in maintaining the infection in nature; the main reservoir is the fox. In Canada and Czechoslovakia the fox also plays a predominant role. In Iran rabid wolves are the greatest danger; the Iran Pasteur Institute reports that from 1949 to 1958 a total of 443 persons were bitten by wolves, with 39 deaths. Elsewhere, the dog plays the most important role. The complexities of the situation are, however, considerable, as is exemplified by the Institut Pasteur of Algeria, which reports the following rabid animals in 1958: 915 dogs, 91 cats, 27 rats, 13 heads of cattle, 10 donkeys, 9 jackals, 4 monkeys, 2 mice, 1 fox and 1 rabbit.

Countries reporting no record of rabies include Basutoland, North Borneo, Guadeloupe, Martinique and New Zealand. No case of rabies in animal or man has been reported in Norway since 1809, in Australia since 1867, in Sweden since 1871, in Denmark since 1889, in Ireland since 1903, in the United Kingdom since 1922, in the Netherlands since 1923, in Belgium since 1930, in Switzerland since 1949, in Singapore since 1953, in Hong Kong since 1955, and in Portugal, Japan and Uruguay since 1956.

Abstracts from Medical Literature.

DERMATOLOGY.

The Pathogenesis of Ringworm.

P. PINETTI (*Panminerva med.*, June, 1959) dismisses the traditional concept that the parasitized hair in ringworm behaves inertly, that is to say that it is invaded by the fungus without exerting its own influence on the invasion. He believes that the fungus has a specific capacity to hydrolyse keratin, that it enters the cortex of the hair within the follicle and proliferates there. However, it does not proliferate in the more distal part of the hair, formed exclusively of structurally and biologically developed keratin (which differs from that in the more proximal part of the hair); here, presumably, essential growth factors are lacking. To maintain the parasitism within the hair which determines the classical picture of the disease, the two growing forces, that of the hair bulb which continually produces keratin and that of the fungus which tends to invade the immature keratin as it is produced, must be perfectly balanced. When the growth of the hair is greater than that of the fungus, the parasitic fungus is soon expelled. The author discusses along similar lines the involution of ringworm of hairs during the period of quiescent hair growth, after bacterial infection and after X-ray depilation.

Antimycotic Cream and Powder.

E. S. BRESTON (*A.M.A. Arch. Derm.*, October, 1959) describes new formulæ for an antimycotic cream and an antimycotic powder. The saturated nine-carbon fatty acid, pelargonic acid, whose antifungal activity equals that of undecylenic acid, was combined with sulphur, because reactivity is not a problem with this combination. The sulphur ("Thylox") was a fine-particle (1μ to 3μ) highly reactive material obtained from coal-gas purification. Two formulations were developed: one was a cream containing 4% of pelargonic acid, 10% of zinc pelargonate and 2% of the fine particle sulphur in a vanishing cream base; the other was a powder containing pelargonic acid, 2·5%, zinc pelargonate, 20%, and zinc sulphide, 3%, in a talcum base. Patients were instructed to apply the cream to the affected areas night and morning. The powder was used in cases of tinea pedis as adjunctive treatment with the cream. In cases of tinea cruris, tinea pedis and tinea corporis treatment with these preparations has given satisfactory results. In infections with *Trichophyton rubrum* improvement rate and relapse rate were higher than for infections due to other organisms. In tinea capitis due to *Microsporum audouini* results have been unsatisfactory. Irritation from these preparations was observed only on mucosal surfaces. No other adverse reactions were observed.

Contact Dermatitis due to Neomycin.

H. G. REYNOLDS *et alii* (*A.M.A. Arch. Derm.*, October, 1959) discuss the clinical features of contact dermatitis due to neomycin. They state that neomycin sensitivity is not common. However, the onset may be insidious, and its recognition depends on a heightened index of suspicion rather than on the appearance of the clinical signs of acute contact dermatitis. A total of 28 patients have been observed with neomycin sensitivity. Illustrative cases are described.

Triamcinolone Therapy for Alopecia Universalis.

W. B. SHELLEY, J. S. HARAN AND J. H. LEHMAN (*A.M.A. Arch. Derm.*, October, 1959) report a striking result obtained with long-term triamcinolone therapy in a case of alopecia universalis. The clinical resurgence of hair growth in the patient appeared within three weeks. Triamcinolone was effective only when given systemically, and topical applications were without effect. In the case under discussion the condition was of prepubertal onset and nine years' duration, and the patient responded well to a low dosage of triamcinolone over a period of one and a half years. However, the steroid effect still remains a completely reversible one, since reduction in the maintenance dose below 6 mg. per day has invariably led to the appearance of alopecia areata lesions. Thus, despite prolonged administration, triamcinolone has failed to have any curative effect. This indicates that the physician must have considerable reservation about suggesting the use of systemic steroids in the treatment of a disease of cosmetic significance only.

Leucocytosis in Dermatitis Herpetiformis.

Z. EVEN-PAZ AND F. SAGHER (*Brit. J. Derm.*, October, 1959) discuss the leucocyte count in three cases of dermatitis herpetiformis. In each of these three cases there seemed to be a relationship between a high leucocyte count of from 37,000 to 68,000 cells per c.mm. and the state of the cutaneous disease. A lymphocytosis of nearly 80% was present in spite of almost continuous and intensive treatment with ACTH or cortisone, which generally lower the lymphocyte count. This picture, with associated enlargement of the superficial lymph glands, and spleen and bone marrow abnormalities, strongly suggested lymphatic leukæmia. However, although immature lymphocytes were seen, truly pathological cells were never detected in the peripheral blood or bone marrow. It is suggested that these should be regarded as leukæmoid reactions. The possible causative role of the disease process or of the drugs used in its treatment is briefly discussed.

Relaxin in the Management of Raynaud's Disease.

H. REYNOLDS AND C. S. LIVINGOOD (*A.M.A. Arch. Derm.*, October, 1959) discuss the use of relaxin in the management of ulceration and gangrene due to collagen disease. The authors have used relaxin in the treatment of two patients who presented with Raynaud's phenomenon and who had associated trophic ulceration and gangrene. These two cases are discussed in detail. The management of the disabling peripheral vascular complications of scleroderma, Raynaud's disease, etc., poses a tremendous therapeutic challenge. No single form of therapy is uniformly effective. The authors state that while relaxin would not be the initial drug of choice, it would appear to be a useful adjunctive measure, and suggest that the combination of relaxin and steroid drugs may be of value in such cases.

UROLOGY.

Spontaneous Remission of Metastasizing Adenocarcinoma of Kidney.

J. D. HALLAHAN (*J. Urol.* (*Baltimore*), April, 1959) reports the case of a 75-year-old man with adenocarcinoma of the left kidney, and local and pulmonary metastases. The pulmonary metastases spontaneously regressed after nephrectomy, and had not recurred two years and nine months later. It has been estimated that complete regression of tumours in general occurs in about one in 100,000 cases. This phenomenon, so far as the kidney is concerned, has been explained by Bartley and Hultquist as a fibrous transformation in conjunction with hyalinization, calcification and ossification. However, in this case no radio-opaque shadows were visible at the site of the previous tumour shadows. An explanation of the fate of tumour emboli lodged in tiny end vessels in the lungs is that they become surrounded with a hyaline thrombosis which in time becomes organized. After this, loss of viability occurs in the tumour cells. The author states that the field of immunology has also been explored for an explanation of such spontaneous changes, but that, so far, preparation of an antigen from the malignant cells of a patient has not produced any promising results. On the basis of this case, the author suggests that nephrectomy may be justified even in the presence of extensive spread of the disease, on the long chance of spontaneous remission of the metastases.

Long Survival with Renal Carcinoma.

C. A. KUEHN AND P. DAVIS (*J. Urol.* (*Baltimore*), April, 1959) state that recent literature is sparse in offering survival rates of patients who have undergone nephrectomy for renal carcinoma. In all available literature over the last five years, the authors were unable to find any cases of long-term survival that have been confirmed by microscopic slides showing the nature of the original tumour and that of the tumour causing death. The case is presented of a male who was 34 years old at operation for removal of a renal carcinoma in 1943, and survived for 14 years. The tumour consisted of largely clear, large polygonal cells, arranged in clusters or masses; no definite tubule or gland formation was apparent; the surrounding parenchyma was compressed, but there was no definite encapsulation; although the neoplasm was highly vascular, no evidence of vascular extension of the growth was seen; mitotic figures were rare. The patient then remained quite well for 12 years, till 1955, when he experienced

dull right flank pain, which gradually increased in severity, and led him to seek advice in 1956. A firm mass was discovered under the old right lumbar scar; there was also a firm mass occupying the entire epigastrium and protruding 4 cm. below both costal margins. The mass was irremovable, and biopsy showed it to consist of fibrous and fatty tissue, partially replaced by a malignant epithelial neoplasm of approximately the same structure as the original renal carcinoma. In spite of X-ray therapy, the patient died just 14 years after his nephrectomy. The original growth had involved about five-sixths of the right kidney, only the upper pole being free, and had extended to the mid-line of the abdomen; a portion of the renal capsule had to be left adherent to the peritoneum.

Ureteric Obstruction from Retroperitoneal Inflammation.

D. J. SHAHEEN AND A. JOHNSTON (*J. Urol.* (*Baltimore*), July, 1959) state that bilateral ureteric obstruction caused by an inflammatory retroperitoneal process is being discovered more frequently in recent years. The ætiology is unknown, and this disease usually appears without any previous history that would suggest its cause. The treatment has not been standardized, but the actual ureteric obstruction must be relieved to protect the kidneys. The authors report two patients who were treated by open exploration, ureterolysis and nephropexy without diversion of the urine or intubation of the ureters. Most patients who have been reported have had a nephrostomy, a ureterostomy, or both. The operation revealed a well-formed sleeve of inflamed periureteric tissue extending from the pelvic brim to the kidneys. The most interesting discovery was that of a band of firm, fibrous tissue, about 0·5 cm. in width, running across the ureter at the pelvic brim. Distal to the band there was no sign of periureteric inflammation. The band consisted of fibrous tissue and thrombosed genital vessels. This finding suggests that the process may begin in the ovarian (or spermatic) veins as a phlebitis, and may then spread retroperitoneally.

Contact Radiation of Vesical Tumours.

E. F. LUTTERBECK (*J. Urol.* (*Baltimore*), July, 1959) states that identical tumour dosages will produce the same biological effect whether we use conventional Röntgen rays, supervoltage radiation, radium or cobalt-60 beams, or any other radioactive source. The chief difficulty is to get the best means of delivering a canceridal dose of an average of 6000*r* to the tumour cells, while sparing the healthy tissues as much as possible. This is frequently complicated by inability to estimate accurately the extent and depth of a given growth. This applies particularly to deep-seated growths such as bladder tumours. The idea of contact X-ray therapy for vesical growths was first described by Pack of New York in 1938. It was developed because it was realized that it is difficult to place radon seeds accurately so as to obtain a homogeneous application of the radiation. The Philips contact tube operates at 50 kilovolts and two milliamperes, with a focal target distance of 20 millimetres. The contact area of the tube is 2·5 cm. Radon seeds and radium or cobalt-60 needles deliver an average of 10 to 60*r* per hour on the surface. The output of the Philips tube is between 6000 and 12,000*r* per minute. There is a rapid fall off of the radiation in deeper layers. The method is applied by the open cystotomy route. The growth is looped off, and then the base is coagulated. The Philips tube is then covered with a sterile condom and brought into contact with all fulgurated surfaces to deliver 6000*r* to the tumour base, which requires an average of 60 to 80 seconds per field. The tube is held by the radiologist over the operator's shoulder and inserted into the open bladder. The hand is protected by a lead-glass shield, and the body by a lead apron. After treatment, the bladder is closed. Twenty-five patients have been treated in the last eight years. A local recurrence in the irradiated area has been seen only once. In two patients there was recurrence of the disease later in other areas. Three patients died from other causes a few months later, and histopathological examination revealed no cancer cells. No disturbing radiation damage has occurred.

Podophyllin in Vesical Papillomatosis.

E. J. RICHARDSON (*J. Urol.* (*Baltimore*), August, 1959) reports the case of a patient with multiple small papillomas accompanying one large growth; these were successfully treated by the local application of a suspension of podophyllin in mineral oil. The patient was a man aged 61 years, and between 1954 and 1957 he had undergone repeated endoscopic resections of the large growth, with coagulation of the numerous small ones, but the papillomatosis could not be controlled. In such circumstances total cystectomy is usually considered necessary, but the author tried out the method of using podophyllin recommended by Duckwater in 1950. This consists of four intravesical injections of increasing strengths (1% to 12%) of the podophyllin suspension at weekly intervals. At the end of such a course all the tiny growths had almost disappeared, but the large growth remained. The second course was given four months later, followed by endoscopic resection of the larger growth. Several check cystoscopies were performed up to one year later without observing any recurrence of the papillomatosis or of the larger growth. This method is of no value in growths which infiltrate the bladder wall, but it is apparently of great value in the typical low papillomatosis which is entirely mucosal.

Neoplasms in Undescended Testes.

C. A. LINKE AND J. H. KIEFER (*J. Urol.* (*Baltimore*), September, 1959) state that, over a period of 30 years, 48 patients with testicular neoplasms were seen at their hospital. Nine of the growths were present in undescended testes. These nine cases were carefully studied. In three of the nine cases the presenting symptom was enlargement of the testis. In one other there was pain in the region of an inguinal testis. In the remaining five the chief complaint seemed, at first, to be unrelated to testicular disease. The histological diagnosis was seminoma in all but one case, in which a surprisingly innocent type of embryonal carcinoma was found. In four of the nine cases non-descent was bilateral; in the remaining five the testis not involved by the tumour was normally descended. From detailed study of this group the authors conclude that, despite recent attempts to minimize its importance, the increased probability of neoplasm occurring in undescended testes is a real and important factor in making a decision as to proper treatment. They consider that any undescended testis which shows enlargement, induration or variation in consistency should be removed and histologically examined, and that all patients who have had undescended testes treated by orchidopexy should have check examinations every six months.

Metastasis in Vesical Carcinoma.

T. R. FETTER *et alii* (*J. Urol.* (*Baltimore*), June, 1959) state that the exact rate of metastasis of bladder tumours is not well established. Although many investigators think that metastasis is late, some reports indicate that metastases may be the source of the first symptoms. The material for this study was gathered from 55 out of 5595 consecutive post-mortem examinations at the Jefferson Medical College, Philadelphia. Metastases were found in 39 of these 55 cases, an incidence of 71%. The most frequent sites of metastases are lymph nodes, liver, lungs and bone, lymph nodes being the structures most frequently involved. The lungs were the most commonly involved parenchymal structure.

Neoplasms of the Renal Pelvis.

W. N. TAYLOR (*J. Urol.* (*Baltimore*), October, 1959) reports on 32 cases of neoplasm arising from the epithelium of the renal pelvis. The age of the patients varied between 32 and 81 years; there were nine females and 23 males. The most frequent presenting complaint was gross hæmaturia. The diagnosis chiefly rests on the observation of filling defects in pyelograms. It is generally accepted that nephro-ureterectomy is called for, regardless of the degree of malignancy, provided the disease is unilateral and that the opposite kidney works well. The intramural portion of the ureter must be included in the excision, otherwise the rate of recurrence of the tumour in the bladder is doubled. Total nephro-ureterectomy was performed on 14 patients in this series, and simple nephrectomy on 13. The prognosis is unpredictable, but is generally poor. There were 31 papillary carcinomas and only one squamous-cell carcinoma. Two patients had had a kidney removed elsewhere, and of the other 30 patients in whom the lesion was studied, only eight have survived more than five years. Five others have survived more than three years, and 17 died in less than three years. Ten of these 17 died from metastatic cancer, one from coronary disease, two from apoplexy, one from pulmonary tuberculosis and two after operation (an operative mortality of 6%).

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on February 17, 1959. DR. W. L. CALOV, Honorary Consultant Physician, was in the chair. The principal speaker was MR. M. INGLIS, Honorary Assistant Surgeon.

Clinical History.

A man, aged 61 years, had suffered from attacks of pain in the region of the right hip joint, radiating to the toes, for three months. Numbness and paræsthesiæ over the anterior aspect of the thigh had also been noted over this period. There were no other relevant symptoms and no history of trauma. There had been no weight loss, chest pain, breathlessness, œdema, hæmaturia or dysuria. Urine was passed twice at night and four times by day. There was no history of jaundice, abdominal pain or melæna. Bowel actions were regular. In the past the patient had suffered from duodenal ulcer for 16 years. X-ray examination of the pelvis six years previously had revealed Paget's disease. A cyst had been excised from the left leg one year previously and there had been one attack of pleurisy.

On examination of the patient, sensation was found to be impaired in the second and third right lumbar dermatomes. There was no restriction of movement of the right hip. A large, firm, tender mass was detected in the right hypochondrium, extending medially to the mid-line and inferiorly to just below the level of the umbilicus. It appeared to move with respiration and there was resonance over the mass. The blood pressure was 130/80 mm. of mercury. Other findings were as follows: the urine did not contain protein or sugar, the hæmoglobin value was 16·3 grammes per 100 ml. and an X-ray examination of the chest revealed no abnormality. X-ray films showed Paget's disease in the pelvis and the soft tissue mass in the hypochondrium.

The provisional diagnoses were ? renal cyst, ? Grawitz tumour. He was subsequently operated upon and later discharged.

Clinical Discussion.

MR. M. INGLIS: It would seem that the salient features of this case are as follows: The patient was a male aged 61. He complained of pain in the right leg for three months. Associated with this was impairment of sensation in the second and third lumbar dermatomes. A large mass was found in the right hypochondrium. A cyst had been removed from the left leg one year previously. There had been one attack of pleurisy, we do not know when. No information regarding rectal examination is given, but one must assume it was performed and findings were normal. I am told a white cell count was not performed.

Let us first consider briefly the cyst removed from his left leg one year before he presented. We are told nothing of the location, nature or possible recurrence of this lesion. Four possibilities exist. Firstly, the cyst in the leg is irrelevant. Secondly, the cyst and mass in the right hypochondrium are both secondary to another lesion. Considering inflammatory lesions as a primary cause, it is unlikely the patient would be unaware of it if an abscess were present. If the two were neoplastic secondary deposits, the most likely primary would be bronchogenic carcinoma, and one would not have anticipated finding a normal chest X-ray twelve months after removal of a peripheral metastasis and with such a large mass present in the hypochondrium. Thirdly, the tumour in the right hypochondrium is secondary to the cyst in the leg. Malignant melanoma is unlikely to be described as a cyst by the patient. A remote possibility is to suppose the cyst was in fact a malignant synovioma. Lastly, the cyst of the leg was secondary to the large mass. Occasionally Grawitz tumours metastasize to the skin, and although any part of the body may be affected, Aird cites the face and scalp as common sites and quotes Scorer as referring particularly to the skin of the lower leg. Of these possibilities, I think the first is most likely; the cyst was incidental.

Next let us consider the neurological symptoms and signs. It is inferred that the pain and the sensory defect were referred. Indeed, in one of the X-rays we can see that the upper half of the right femur appears normal. In the plain X-ray of the abdomen there is little evidence of osteoarthritic changes and no evidence of metastases, but I think a lateral and oblique view of the lumbar spine would have been reasonable to demonstrate better the intervertebral foramina. We are not told of the motor power, nor of the presence or absence of quadriceps wasting, nor the state of the deep reflexes. I think we must assume these signs and symptoms were due to pressure on, or infiltration of, the nerve roots or plexus.

Now let us turn our attention to the mass in the right hypochondrium. Locating its site is the first step in diagnosis. Although the described physical signs leave us in some doubt, the X-rays show the kidney high under the diaphragm on the top of the mass, and the colon appears to be lying in front of it, thereby suggesting a retroperitoneal lesion, and I shall not consider lesions of liver or gall-bladder. The barium enema excludes a carcinoma of the hepatic flexure, and although a poorly filled diverticulum of the ascending colon remains as a possible site of inflammation, the absence of abdominal pain, the normal temperature, the absence of a hot bulge in the loin, so far as we are aware, make an abscess unlikely. The lack of abdominal symptoms or jaundice I think rules out lesions of the head of the pancreas or porta hepatitis. The big question which remains is whether or not the mass arises from the lower pole of the kidney or below it. I do not think we can say with any certainty.

DR. W. L. CALOV: Would you like Dr. Williams to demonstrate the X-rays now?

MR. INGLIS: I think that would be a good idea.

DR. WILLIAMS (pointing in reference to the intravenous pyelogram series, a chest film and a barium meal): This shows the mass in its greatest density and in relief against the background. That is the extent of the very smooth spherical mass. Above it is the kidney with slightly dilated pelvis, calyces also a little dilated, riding up on top of this very big mass. The Paget's disease you can see. There is no evidence that a sarcomatous transformation has occurred. The small bowel and transverse colon are obviously in intimate relations with the mass, and are displaced down and follow to some extent the inferior edge of this cystic swelling. There are no obvious vertebral changes, and we have almost a lateral film which does show us the foramina of the second, third, fourth and fifth lumbar vertebræ without obvious expansion of the openings. There is a certain amount of osteoarthritic change not very consistent with his age, a little lipping and minor senescent changes with disks a little narrowed generally. The psoas margin is not defined on the right side, but is beautifully clear on the left, and I think the bulk of the mass itself would be enough to obscure the psoas muscle in the antero-posterior film.

MR. INGLIS: If the mass is arising from the kidney, a simple renal cyst and Grawitz tumour are the two most obvious diagnoses. A hydatid cyst of this size without liver or lung involvement is unlikely, and for the reasons already given I do not think this mass is in the liver. A secondary sarcoma in the kidney is also a possibility, but a primary must be postulated. Apart from the cyst removed from the leg, this man has had Paget's disease for at least six years; but there is no evidence of sarcomatous change in the Paget's disease as we see it there. I have already ruled as unlikely a secondary carcinoma of the lung, and there is nothing to suggest carcinoma elsewhere.

In favour of a renal cyst is the continuing reasonably good function in the kidney on that side. There is dye in the first of the intravenous pyelogram films. A Grawitz tumour could, however, reach the size of this mass, yet leave the function largely undisturbed.

The neurological signs are more consistent with a diagnosis of Grawitz tumour, producing them either by infiltration of the roots or by metastasis in the lumbar vertebræ. The question of cyst or Grawitz tumour would be resolved by a successful aortogram; but in view of its size, operation would be advisable in either event, and since aortography has a mortality, though small, I do not think it would be justified in this case.

I think that of the two, Grawitz tumour is the more likely.

If this tumour is below the kidney, and in some of these films one may gain such an impression, a retrograde pyelogram would help to evaluate the problem of whether or not it arises from the lower pole of the kidney or from below the kidney and is displacing it upwards.

Now let us consider the possibilities, supposing it lies below the kidney. It is unlikely to be a retroperitoneal teratoma, as I believe these rare tumours occur only in women. More probable is a retroperitoneal sarcoma. It may be a leiomyosarcoma or a rhabdomyosarcoma, though

the size of the mass and the age of the patient make the latter very unlikely. A malignant schwannoma, liposarcoma and lymphoma are further possibilities. One physical sign is very much against these lesions as the probable diagnosis; this is mobility of the mass with respiration.

With a mass reaching so high, it is possible that a liposarcoma might move on respiration, as it is not tethered from its inception as the other sarcomata mentioned usually are, and also since it is the commonest of these rather rare tumours of the retroperitoneum I shall make this my diagnosis amongst the retroperitoneal growths.

I believe that the most important step in diagnosis is missing—namely, ascertaining for certain whether this mass arises from the kidney or from below it. If the reconstruction of the case as I have made it is correct, I feel that, confronted by this case in the wards, one would say "This is probably a Grawitz tumour", and I think I would add, "Let us have a retrograde pyelogram with some lateral views".

I am, however, influenced by the fact that this is given to us in the protocol, and yet the case has come to a clinico-pathological conference. I shall therefore make as my diagnosis, for I think one should be made at these meetings, a retroperitoneal sarcoma, probably liposarcoma.

DR. CALOV: The case is now open for discussion. Would you care to make further comment on the X-ray films, Dr. Williams?

DR. WILLIAMS: There is a projection from the caecum at its junction with the ascending colon, a curious tongue-like distorted shadow which could possibly be the terminal ileum itself; but I am not convinced it is. There is also this very pretty smoothing-out impression on the small bowel, and one cannot completely ignore the possibility of a reduplication cyst of the midgut, although it is rather posterior, and I cannot see the appendix with any certainty and a history of its removal is not given, so once again one cannot exclude a lesion of the appendix itself which may occur in this position.

DR. L. WATSON: I would like to say a word in favour of hydatid cyst as a real possibility. Mr. Inglis pointed out that such a large hydatid cyst occurring alone is a rarity, with which I entirely agree, but I think we must always think of it seriously when we have a large round cyst of this kind in this country. I am remembering a similar meeting to this some years ago, where the case of a space-occupying lesion in the cranium was localized by the neurologists, but its nature was missed completely, and hydatid was not seriously considered because of lack of other evidence of it. The pathologist on this occasion produced a hydatid cyst which had been shelled out at operation.

MR. I. POTTS: I have unfortunately arrived a little late, and so would like to comment only on the latter part of the discussion.

It must be rememebered that as far as Grawitz tumours are concerned, 90% present with the classical triad of pain, hæmaturia and a mass, which may be a considerable help to clinical diagnosis. This man was found to have a mass, which evidently he was not complaining of, but in the protocol we find it was tender. The appearances of the films (from this distance) favour a cystic lesion; but I cannot see the collecting system with any clarity. The urologist is frequently faced with the problem of X-ray diagnosis of cyst versus tumour. From a large experience at the Mayo Clinic, Emmett and Brasch considered that there are no distinctive differentiating features, and that accessory evidence is required.

This accessory evidence is obtained either from perirenal air studies or by aortography. Briefly, with an air study, the air should readily surround a cyst and give a clearly-defined outline in the X-rays. With the aortogram, if it is the ordinary vascular type Grawitz tumour, then increased vascularity of the kidney and the tumour will be shown, whereas, if it is a cyst, an avascular area should be seen. These further studies may make the diagnosis obvious.

Some would consider that a tumour presenting so well in the loin as this, should be needled to see whether the contents are fluid or not, and then some dye injected to obtain a cystogram. The obvious objections to this procedure are the possibility of puncturing a neoplasm and producing bleeding or implantation along the needle tract.

Hydatid of the kidney is very rare indeed. At a meeting at Royal Prince Alfred Hospital some years ago, only four had been seen in 25 years. I should personally imagine a hydatid of the kidney of that size would have some calcification of the periphery.

On the X-ray films as they stand, the diagnosis between cyst and carcinoma cannot be made.

MR. H. H. PEARSON: Fortunately, or unfortunately, I remember the case, and so my remarks may be prejudiced. However, in my experience it is rare to get dilatation and distortion of the calyces of this nature with an extra-renal lesion. An extrarenal mass leaves the ureter and collecting system displaced but more normal in appearance, so on the films alone it is more likely to be renal.

MR. A. SHARP: If Mr. Inglis thinks these neurological signs are due to infiltration, what does he think of the hæmoglobin value of 16·3 grammes?

MR. INGLIS: It is high.

DR. C. S. H. REED: One point in regard to hæmoglobin level is that there are a number of retroperitoneal growths associated with polycythæmia. I mention this as an isolated fact, not seriously for discussion.

In view of this man's admission 12 months ago for excision of a cyst of his leg, one would presume that his abdomen was examined at this time, and as the mass was not found then, it may have arisen fairly rapidly. There is no weight loss. This is significantly against a rapidly expanding neoplasm, and a point, I feel, in favour of this mass being a cyst which has expanded rapidly without causing much pain or constitutional symptoms. I would like to support the idea of a renal cyst.

DR. CALOV: Any further questions? What about the students?

DR. H. M. WHYTE: On behalf of the students, I would like to ask what the physical features of a renal mass are.

MR. I. POTTS: The classical physical signs of a renal swelling are these. Firstly, it arises in the loin, and, if mobile, can be pushed back into the loin more readily than elsewhere. It is reniform in shape in the early stage. It moves with respiration unless fixed by inflammatory or neoplastic adhesions in the perinephrium. It is dull to percussion anteriorly and posteriorly, except for the band of colonic reasonance which may be elicited anteriorly. When large, it may be ballotted between a hand in the loin and one on the flank. Very, very rarely, with a kidney full of stones, one may feel calculus crepitus.

DR. B. P. BILLINGTON: What sort of cysts are there on the leg?

MR. INGLIS: One really requires notice for this question. There are not really many. Most of the sebaceous cysts one sees are not on the leg. I think that there should have been some attempt made to find out what this cyst was; no doubt there was. We could have been told in the protocol whether the scar was in relation to the knee joint, when it might have been a ganglion or bursa.

Pathological Report.

DR. A. A. PALMER: The specimen received was a right kidney, grossly enlarged, measuring 18 by 13 by 9 cm. The upper two-thirds of the kidney appeared normal. The lower pole was replaced by a large globular cyst 13·5 cm. in diameter. The external surface of the cyst was smooth. The contents of the cyst were dark brown fluid and shreds of yellowish material. The wall was 0·5 cm. thick, fairly uniform and apparently fibrous. The cut surface of the upper part of the kidney was normal, as was the lining of the pelvis.

Microscopic examination of the cyst wall shows compressed and fibrous renal tissue and areas of clear cells, with some more solid islands of malignant cells in places invading the renal veins. This is characteristic of a Grawitz tumour; the growth is unusual, in that it has undergone rather extensive central degeneration and hæmorrhage, which latter feature may have been responsible for the apparent rapid increase in size.

Some statistics regarding these tumours are quoted from E. T. Bell. Of Grawitz tumours, 95% occur over the age of 40, and the peak incidence is 60 to 70 as in our case; 70% occur in males. A very distinct relation exists between size and metastasis of the tumour; in Bell's series, of those under 3 cm. in diameter, only 2% had metastases; but taken all together, including all sizes, 50% had metastases at the time of diagnosis. Venous invasion to the extent of reaching the inferior vena cava was present in 13% of Bell's series.

A large series of 500 cases was reported by J. T. Priestley in 1939. He had 48% three-year survival, 38%

five-year and 27% ten-year survivals. It is of interest that even some of those in whom the tumour extended into the renal vein survived 10 years, and further, that occasional patients in whom a metastasis was resected from a lung or a bone had long survival.

Diagnosis.

Grawitz tumour.

British Medical Association.

VICTORIAN BRANCH: SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on November 12, 1958, at the Medical Society Hall, 426 Albert Street, East Melbourne. The meeting took the form of a symposium on neonatal problems.

Neonatal Problems.

Jaundice.

DR. T. G. MADDISON read a paper on jaundice. He said that jaundice was a common problem in the neonatal period, as nearly all premature infants and some mature infants became jaundiced. It was important to know when the jaundice first developed, for how long it persisted and how deep it became, as a consideration of those time and intensity factors would help the diagnosis and management. One gramme of hæmoglobin would form 35 mg. of bilirubin. That breakdown took place in the reticuloendothelial cells, and the bilirubin formed was present in the plasma and extracellular spaces, adsorbed to albumin. That form of bilirubin gave the indirect diazo reaction, was the type usually found in the neonatal period and could, in high concentrations, damage the basal ganglia, producing kernicterus. Conjugation of the bilirubin with glucuronic acid occurred in the liver cells. That enzyme reaction produced the soluble directly-reacting bilirubin which was excreted. In certain rare obstructive lesions, the direct bilirubin might be present in the plasma. The inefficiency of that enzyme reaction was responsible for most cases of neonatal jaundice, and in particular for the jaundice of premature infants. In general, the smaller the premature infant, the deeper the jaundice. It usually developed on the second to fourth day and might last for a week or more. The bilirubin level usually rose until the fourth or fifth day and then fell, presumably as the efficiency of the liver enzyme system improved. Any associated condition, such as pulmonary atelectasis, infections, anoxia or bruising, would increase the depth of the jaundice. Obviously any premature infant required good general care and attention to the fluid requirements. Recent work had also drawn attention to the dangers of giving too much vitamin K. As a prophylactic dose, 0·5 to 1·0 mg. was sufficient. A dose of 30 mg. (contained in three millilitres of "Synkavit" solution) would increase the bilirubin levels of premature infants. It was best to clamp the cord immediately after delivery. The extra placental blood (which might amount to three ounces) would only lead to further bilirubin production in the next few days. Exchange transfusion could be used to lower the bilirubin levels in jaundiced infants. In theory it was easy. An exchange transfusion was given when the indirect bilirubin level was above 20 mg. per 100 ml., or when it was obvious that it would rise above 20 mg. per 100 ml. There was a large extravascular store of bilirubin, and eight to twelve hours after an exchange transfusion the level would reach, or almost reach, the pretransfusion level from redistribution. It might soon be possible to treat deep jaundice by improving the enzyme system in the liver.

Dr. Maddison went on to say that the next common cause of jaundice was blood-group incompatibility. In general, any jaundice which developed within the first 24 hours was due to blood-group incompatibility until proved otherwise by blood tests. The Rh blood-group incompatibility was now well known, as was the need to group the blood of all mothers during pregnancy and perform antibody tests during pregnancy if they were Rh-negative. The clinical picture in the infants might vary from mild anæmia to severe hydrops. Typically the enlarged liver and spleen, the anæmia and the positive response to the Coombs test in association with early jaundice would establish the diagnosis. Exchange transfusion had materially changed the prognosis for severely affected babies who were born alive. Interest was now being centred on trying to salvage the babies previously lost by fœtal death in utero. Success in those pregnancies depended on the cooperation of many people, and the time for induction of labour was a difficult decision for the obstetrican to make, even with all the available facts. Dr. Maddison then quoted a case to illustrate some of the difficulties. The mother had been immunized by a previous blood transfusion, and during her third pregnancy the antibody titre rose steeply to very high levels. Labour was induced six weeks before term. The baby weighed 5 lb. 7 oz., and had a large liver and spleen; the hæmoglobin value of the cord blood was 50%. He required three exchange transfusions within the first 48 hours, as the serum bilirubin level rose to 22·5 mg. per 100 ml. at 18 hours, and to 23 mg. per 100 ml. on the second day.

Dr. Maddison then said that A B O incompatibility was the other common blood-group incompatibility. It was really just as common as Rh incompatibility if looked for. In general, the clinical features in the infant were milder. The clinical features in most instances different from those in the Rh-immunized infant, were: (i) often it was the mother's first pregnancy; (ii) there were no stillbirths; (iii) jaundice was of early onset; (iv) the anæmia was mild; (v) splenomegaly was slight; (vi) usually early recovery occurred; (vii) kernicterus might occur. The main laboratory findings were: (i) the mother belonged to blood group O; (ii) the baby was of group A1 or B; (iii) the direct Coombs test produced a negative result; (iv) the maternal anti-A or anti-B titre was often high; (v) reticulocytosis and spherocytosis were present; (vi) the indirect bilirubin level in the serum was elevated. After affected infants were recognized, they were watched carefully, especially in relation to the serum bilirubin level. An exchange transfusion might be necessary if the serum bilirubin level rose above 20 mg. per 100 ml. That was not often necessary for full-term infants, but might be necessary for premature infants.

Dr. Maddison then said that there remained only the rare causes of jaundice, which could be grouped under four headings: infective, surgical, metabolic, and miscellaneous blood conditions. Ascending bacterial infections from the cord were now fortunately rare causes of liver damage and jaundice. Certainly in Melbourne non-suppurative hepatitis was the least rare infection. It appeared that the virus of homologous serum jaundice was the cause, and was transmitted across the placenta in mothers who were "silent" carriers. The clinical picture was one of the early onset of jaundice in the first week or weeks of life, acholic stools, bile-stained urine, moderate hepatosplenomegaly, and a high percentage of direct bilirubin in the plasma. It was in those cases that the differential diagnosis from bile-duct atresia was difficult and sometimes impossible without surgical exploration. Fatal hepatic necrosis could be caused by the herpes simplex virus. The source of infection might be genital herpes in the mother or oral herpes in an attendant. That fact should be emphasized so that such persons were excluded from nurseries for new-born infants. The clinical picture was one of insidious onset several days after birth, with tachycardia and pyrexia often associated with jaundice. Respiratory and hæmorrhagic symptoms developed in association with a rapid terminal decline and sudden enlargement of the liver. Generalized cytomegalic inclusion disease was another virus infection due to the salivary gland virus. In the first week of life the clinical features were jaundice in a premature infant, enlargement of the liver and spleen, purpura with associated thrombocytopenia and hæmorrhagic symptoms. Congenital toxoplasmosis was a protozoal infection derived from the mother. The congenital disease again might simulate erythroblastosis fœtalis in the early days, but chorio-retinitis, intracranial calcification, hydrocephaly or microcephaly developed in the ensuing weeks. The diagnosis could be made by demonstrating the parasites in the tissues or body fluids and by the dye test or complement fixation test on the infant's and mother's serum.

Dr. Maddison said that the surgical conditions causing obstructive jaundice were not common. Atresia of the bile ducts was the most important, and unfortunately only 20% of the patients were operable. Clinically, the jaundice did not appear until late in the first week, and it progressively deepened. After evacuation of the meconium, the stools became greyish-white in colour and the urine deeply bile-stained. Surgical exploration was indicated after various tests had excluded other causes, and even with all laboratory aid the diagnosis from hepatitis was not always certain. Congenital choledochal cysts (or cysts of the common bile duct) were usually found in older children. The classical triad was an abdominal mass, obstructive jaundice and abdominal pain. Rarely a cyst or tumour near the common

bile duct might cause obstructive jaundice by external pressure. One interesting association which had been described was that of obstructive jaundice with congenital pyloric stenosis, an unusual angulation of the pyloric tumour causing pressure on the common bile duct sufficient to produce jaundice in addition to the signs and symptoms of pyloric stenosis. Pigment stones in the common duct had also been described, usually as a complication of hæmolytic disorders.

Dr. Maddison went on to say that of the metabolic disorders, galactosæmia was the most interesting. It was an hereditary inborn error of metabolism due to an enzyme deficiency. The infants became jaundiced in the first week of life, often vomited and always failed to thrive. The liver enlarged as cirrhosis developed. A reducing substance, galactose, could be found in the urine, and they might develop cataracts. Dramatic improvement and normal development resulted from a galactose-free diet. Congenital familial non-hæmolytic jaundice had been described by various authors. It was a specific, permanent defect of the enzyme system necessary for the conjugation of bilirubin. The indirect bilirubin accumulated, and the patients might die from kernicterus in the neonatal period. Scandinavian authors had pointed out an interesting association between cretinism and prolonged neonatal jaundice; the latter preceded the clinical picture of cretinism, which was usually not present until at least the age of three months. That prolonged jaundice persisted for six to 18 weeks. All laboratory tests gave negative results, and the condition might divert attention from the developing picture of congenital myxœdema. Persistent jaundice and hepatosplenomegaly had also been described in thyrotoxic infants born of thyrotoxic, exophthalmic mothers, and also in Niemmann-Pick disease.

Dr. Maddison finally said that amongst the rare blood conditions were congenital spherocytosis and congenital leukæmia. The so-called "bile-inspissation" syndrome might follow in a small percentage of erythroblastotic infants. That presented the picture of obstructive jaundice, and the patients usually recovered. The exact pathology was in dispute. Although there were many rare causes of jaundice, the majority were due to liver immaturity or blood-group incompatibility, the common causes.

Cyanosis.

DR. M. J. ROBINSON discussed cyanosis. He said that it was a basic physiological fact that cyanosis was dependent on the absolute amount of reduced hæmoglobin in the capillary blood. The possible mechanisms capable of producing it were: (i) defective pulmonary ventilation, (ii) the shunt of desaturated blood from the right side of the heart to the left, (iii) an increased quantity of circulating hæmoglobin, (iv) the presence of an abnormal blood pigment and (v) peripheral capillary stagnation.

Discussing first defective pulmonary ventilation, Dr. Robinson said that it might in turn be due to failure of normal lung expansion. Pulmonary atelectasis might result from (*a*) blockage of the respiratory passages with mucus, amniotic fluid, meconium, etc., (*b*) the presence of a semi-rigid thoracic cage and weak muscles of respiration (particularly seen in premature babies), (*c*) inadequate stimulation from the respiratory centre, (*d*) "premature" lungs. It could readily be seen that difficult and prolonged labours and prematurity would frequently be associated with pulmonary atelectasis, and therefore, if severe enough, with cyanosis. The clinical picture of atelectasis was one of dyspnœa with or without cyanosis, which might be intermittent or continuous. The infant was distressed, often sweating and restless, and breathing rapidly. The breathing tended to be short and grunting, with chest retraction. The chest retraction was intercostal, sternal and sometimes supraclavicular. Auscultation revealed deficient aeration and the presence of moist sounds over the affected area or areas. In general the condition of those infants was improved by suction—adequate but gentle clearing of the nasal passages and naso-pharynx. That should be carefully and adequately performed at birth, and was indicated when such a condition was suspected in the neonatal period. Oxygen, preferably given in a humidified atmosphere, was indicated also, and the infant should be nursed in an "Insulcot". A humidified atmosphere prevented desiccation of secretions and excess loss of fluid from the body. The infant was also under constant vision. Dr. Robinson made a plea for radiological examination of the sick neonate with cyanosis, when the diagnosis was in doubt. He said that clinical signs were notoriously unreliable in the neonatal period, and it was vital that an accurate diagnosis be made. Two conditions simulating atelectasis were worthy of mention: hyaline membrane and pulmonary hæmorrhage. In hyaline membrane disease, the alveoli were lined by acidophile, homogeneous granular membrane and extensive atelectasis and intense congestion of capillaries were present. The cause of the hyaline membrane was not yet certain, but several theories existed. It occurs mainly in premature infants, in babies delivered by Cæsarean section and in infants born of diabetic mothers. Clinically, dyspnœa with gross chest retraction and cyanosis occurred and rapidly increased. There was poor air entry over the chest, and fine crepitations could be heard everywhere. Radiologically, a fine reticular pattern could be seen throughout the lungs. In pulmonary hæmorrhage, cyanotic attacks and dyspnœa occurred, and terminally the passage of frothy blood from the mouth and nose. It was a common mode of death in kernicterus, but could often occur apart from that. The clinical picture was very similar to that of hyaline membrane disease. Cyanosis due to defective ventilation might be cerebral in origin. That implied damage to the respiratory centre. As had been mentioned earlier, atelectasis was a common association. The respiratory centre might be damaged by anoxia, drugs, prolonged anæsthesia or cerebral hæmorrhage, and in those circumstances the clinical features of cerebral injury plus respiratory symptoms were present. The latter might predominate, and the signs might thus be mainly cyanosis and abnormal respiration. The breathing might be irregular and shallow with apnœic phases. A cerebral cause should always be considered if the chest signs did not appear to explain the cyanosis, and further evidence of cerebral damage should be sought. The neonate—and in particular the premature infant—who suddenly developed cyanotic attacks and profound respiratory depression should also be remembered; many had an intracerebral and ventricular hæmorrhage.

Dr. Robinson went on to say that defective pulmonary ventilation and cyanosis might result when the lung surface available for aeration was reduced by (i) air and (ii) abnormal intrathoracic contents. There accurate diagnosis was vital, because treatment, and curative treatment, might be instituted. Intrapulmonary cysts both congenital or acquired (the result of infection) occurred, and produced dyspnœa and cyanosis. Rupture might produce a pneumothorax and increase the distress. The clinical signs were a full chest, defective air entry, hyperresonance over the affected side, and displacement of the mediastinum away from the affected side. The exact diagnosis was made radiologically, and that, of course, would determine the exact treatment. A similar picture would be found in diaphragmatic hernia; in that condition dyspnœa, cyanosis and an emphysematous chest were present, with a flat or scaphoid abdomen. Displacement of the mediastinum and deficient air entry over the side involved would make the diagnosis suspect. Again, radiological examination would establish the diagnosis. Next, defective pulmonary ventilation might be associated with stridor and cyanosis. As a rule, stridor in the neonatal period was mild, not associated with cyanosis, and the result of neuromuscular incoordination and abnormally large false vocal cords. It would, however, be tragic if a cyst in the pharynx or lingual thyroid was missed because of failure to look. Should the symptoms be severe and cyanosis worrying, the clinician should always inspect the neck, radiologically examine the chest for a mediastinal mass and consider seriously the question of laryngoscopy. Dr. Robinson said that the picture of œsophageal atresia was now well known, and he could only plead for early diagnosis, when operative treatment would be satisfactory in a large proportion of cases. The clinical picture was that of a baby born of a hydramniotic mother, who was very "mucousy" and developed cyanotic attacks. The diagnosis was certain if a firm rubber catheter failed to pass into the stomach. Radiological examination outlined the upper pouch. It should not be forgotten that the new-born baby might be cyanosed if the nares were blocked. Virus infections and the use of reserpine in the mother were likely to cause that. The syndrome of choanal atresia implied failure of canalization of the nares, proved by failure to pass a catheter. Those babies maintained their colour when an oral airway was arranged, but otherwise rapidly died of asphyxia. Babies born with a maldeveloped mandible (micrognathia) often had a cleft palate and a tongue displaced backwards. That was the syndrome of Pierre Robin. The falling back of the tongue produced cyanotic attacks. The treatment was basically that of arranging a suitable posture, but it was a difficult problem and the mortality was high. The problem of neonatal pneumonia was not a rare one. The syndrome might follow atelectasis, and it might be associated with inhalation, as

in the sick neonate or premature infant. Probably the commonest organism was the *Staphylococcus*. Cyanosis was by no means the common presenting symptom. However, it occurred particularly in the fulminating case or when such complications as obstructive emphysema, pneumothorax or broncho-pleural fistula arose.

Dr. Robinson went on to say that cyanosis in congenital heart disease implied a right-to-left shunt. The infant might appear reasonably comfortable despite, at times, the most intense cyanosis. Cyanosis might, of course, be mild and variable, so that it might sometimes be difficult to detect. As a rule there was no gross chest retraction or sweating or the anxious expression seen in the pulmonary cyanotic group. Oxygen did not, nor could it, relieve the cyanosis in that condition, and the cyanosis was accentuated by crying or breath-holding. The cyanosis was central and the extremities were warm. The absence of a heart bruit in the immediate neonatal period did not mean much; often some 10 to 14 days or more elapsed before the bruit was heard. That was a common finding in mongoloid babies with congenital heart disease. The heart rate was often slower than normal, and that point should be noted. Radiologically the heart might be enlarged or abnormal in outline. The lung fields might be congested, as in transposition of the great vessels, or clear, if pulmonary stenosis was present. The electrocardiogram gave additional information. Other forms of heart disease would produce cyanosis when congestive failure supervened. That might be seen soon after birth. There cyanosis was associated with dyspnœa, cardiac enlargement and in particular enlargement of the liver. Such conditions as myocarditis, fibroelastosis and abnormality of the coronary vessels might present in that way. Cyanosis might be peripheral, and it was not rare to be presented with a neonate whose cyanosis was due to capillary stagnation. That was seen in babies with subnormal temperatures, and in babies who were in peripheral circulatory failure due to dehydration or profound toxæmia from a severe infection (a late cause). Hypothyroidism, rarely diagnosable before three or four weeks, tended to produce that type of cyanosis. As had been mentioned earlier, cyanosis was dependent on the absolute amount of reduced hæmoglobin present. Infants with polycythæmia readily reached that level and thus were commonly cyanosed (e.g., syndrome of placental insufficiency). They might have hæmoglobin levels of 180% or higher. Finally, the presence of an abnormal blood pigment readily produced the picture of central cyanosis; the pigment was methæmoglobin. There were a rare congenital variety and an acquired form. In the neonatal period, the latter tended to result from absorption of aniline dyes, used as marking ink. The cyanosis in both groups disappeared with the use of methylene blue—temporarily only in the congenital variety. Dr. Robinson finally said that if the basic causes of cyanosis were kept in mind, a correct diagnosis in the particular case could usually be made.

Pallor.

DR. W. H. KITCHEN discussed pallor. He said that, influenced no doubt by undergraduate training, many doctors were still inclined to consider asphyxia pallida as the only entity likely to be encountered. Yet in the last decade increasing emphasis had been placed on the occurrence of profound anæmia presenting at the moment of birth. That anæmia, if recognized promptly, could be successfully treated, but if management was poor, death commonly resulted. The ætiology of pallor in the new-born infant fell naturally into one or other of two groups: (i) cerebral birth trauma, in which the pathological basis might be cerebral hæmorrhage or cerebral hypoxia; (ii) severe anæmia from hæmolysis due to Rh and other blood incompatibilities or from blood loss, or of the congenital hypoplastic type. That the group of anæmic infants did contain some rare conditions did not detract from its importance, and greatly added to its interest. It was well appreciated that cerebral hæmorrhage and cerebral hypoxia in their extremest form produced the clinical picture of asphyxia pallida—a pale infant with absent muscle tone and often smeared with meconium. The heart rate was typically slow, and respirations initially were absent, then started with sporadic gasps, and might take up to half an hour to become regular. Often death supervened, perhaps fortunately. Cerebral hæmorrhage in those desperately ill infants was from a ruptured vein of Galen or a severely lacerated tentorium cerebelli which had in turn laid open a lateral sinus. Labour was usually precipitate, or had been terminated by a high forceps delivery or perhaps a breech extraction. In such deliveries, intolerable internal stresses from sudden moulding of the head tore veins and tentorium. The resultant large intracranial blood collections affected the function of the medullary centres. Thus respiration was delayed in onset, and vascular tone was altered to yield the clinically obvious feature of pallor. Such extreme injuries were usually incompatible with survival, and were not amenable to even the most heroic therapeutic measures. Cerebral hypoxia might yield a similar clinical picture when transfer of oxygen to the fœtus was interrupted for a prolonged period. Likely causes were placenta prævia, premature separation of the placenta, suffocative maternal anæsthesia, accidents to the cord such as prolapse, rupture or stretching, or pressure of the presenting part. The fœtus usually indicated its distress in utero by (a) variations in heart rate—very rapid, very slow or irregular, (b) tumultuous fœtal movements, (c) the passage of meconium into the liquor. The final picture from severe anoxia or severe intracranial hæmorrhage might be "asphyxia pallida"—a brain-damaged, pale, limp, almost lifeless child.

Turning to the anæmic causes of pallor, Dr. Kitchen said that the essential features in that diagnostic group were (a) pallor associated with a low hæmoglobin level, (b) a ready revival after delivery, (c) rather rapid, shallow respiration, (d) an initial impression of the baby's being "not too bad" despite a pale, translucent appearance. That apparent good health was likely to be short-lived and illusory; such babies often deteriorated rapidly and died if an expectant policy was adopted. Hæmolytic anæmia in that age group had been discussed earlier in the symposium. It remained to be pointed out that at the moment of birth, jaundice was absent. The mother's circulation very readily disposed of the baby's excessively produced bilirubin. A severely Rh-affected baby might thus present solely with pallor. Associated features, if looked for, would usually supply the clinical evidence of hæmolytic anæmia. Such features included the following. (i) The umbilical cord was often stained yellow, rather than appearing pearly white (care was required to avoid confusion with iodine applied later to the cord). (ii) The vernix might be yellow, not white. (iii) A tendency to œdema might be noted—the appearance of the baby suggested a miniature Cushing's syndrome. (iv) Hepatosplenomegaly was present. (v) The hæmoglobin value in the cord or peripheral blood was low. There remained the heterogeneous group of blood loss; the source might be the bowel, the cord, incision of the placenta at Cæsarean section, ruptured vasa prævia, rupture of placental vessels, or concealed hæmorrhage. The site of blood loss was often obvious; occasionally hæmorrhage was truly concealed, and it might be a mystifying illness. Bleeding from the bowel as a manifestation of hæmorrhagic disease of the new-born, and bleeding from a ruptured or insecurely tied cord required no comment. A not uncommon cause of fœtal bleeding at Cæsarean section occurred when an anteriorly situated placenta was incised at operation and the fœtus lost some of its precious blood. A ruptured vasa prævia might cause sudden severe blood loss. There the cord had a velementous insertion into the placenta, and during parturition an unsupported vein might rupture. That accident was strongly suggested by the loss per vaginam, late in labour, of five or six ounces of bright blood. That occurrence should warn the obstetrician of the possibility that the infant might be severely exsanguinated and in urgent need of transfusion. A surface vessel on the placenta might rupture, with loss of fœtal blood from a site which was truly concealed until the placenta was delivered and examined. The fœtus might lose blood into its own substance, as (a) in a large cephalohæmatoma, (b) in a large subcapsular hæmorrhage of the liver, or (c) from very extensive bruising over the head and neck. Fœto-maternal transfusion was a rare but interesting condition. The baby was born anæmic, and it could be proved that a large fraction of the infant's blood had escaped into the mother's circulation. Fœtal hæmoglobin, which differed biochemically from adult type hæmoglobin, was slowly removed from the mother's circulation in the months after delivery. Twin-to-twin transfusion might be recognized if the obstetrician was alert. In that decidedly rare occurrence, one member of a set of twins with a common placenta was anæmic, the other plethoric. A transfer of blood from the circulation of the anæmic to the plethoric twin could be proved under favourable circumstances.

Dr. Kitchen then said that in considering the loss of blood from the new-born infant, one must realize the real significance of the small volumes involved. It should be remembered that the circulating blood volume of a seven-pound infant was approximately 10 ounces, and that of an adult of the order of 10 pints. An ounce of blood lost to the baby was therefore equivalent to a pint loss from an adult. Thus one must heed blood losses of two ounces or

more and regard them as significant and serious. A unique feature of bleeding in the newborn was the very rapid fashion in which hæmodilution took place. Thus a peripheral blood hæmoglobin estimation was a good and rapid index of the amount of blood lost. That was in sharp contrast to the adult, in whom hæmodilution after hæmorrhage took many hours. It was mandatory to estimate the hæmoglobin value of the pale baby. It was also essential to realize that in the new-born infant the lower limit of normal was 18 grammes per 100 ml. Thus a hæmoglobin level of "100% or thereabouts" represented a definite and significant anæmia. Other investigations include A B O and Rhesus typing of the baby's blood, a direct Coombs test, and possibly a lumber puncture if asphyxia pallida was the likely diagnosis.

Dr. Kitchen finally said that treatment depended on the correct diagnosis. For severe blood loss prompt transfusion was required. Often O4 Rh-negative blood must be used without the precautions of typing and cross-matching. If the problem was a little less urgent and prompt pathological help was available, transfusion in a more careful leisurely fashion might be carried out. The umbilical vein was large, readily identified and a good site for an emergency transfusion. The management of the erythroblastotic infant had been considered earlier by Dr. Maddison. In asphyxia pallida the therapy was often unsatisfactory, because of the serious nature of the baby's injury; the child was born virtually dead, and frequently (perhaps fortunately) oxygen, chemical stimulants and even lumbar puncture failed to revive him.

Discussion.

Dr. Robinson, in reply to questions regarding the use of oxygen, the hæmoglobin level at which transfusion was indicated and the use of vitamin K, said that there was no doubt that in premature babies large amounts of oxygen caused retrolental fibroplasia. If the infant was grossly distressed it was essential to use oxygen, but it should be stopped when the colour was satisfactory. The use of blood transfusion was not governed by any particular level of hæmoglobin, and he was guided largely by the clinical condition of the child. A prophylactic dose of 0·5 to 1·0 mg. of vitamin K was adequate, and if bleeding was occurring, 5 mg. was more than adequate. It was questionable whether larger doses were of any value.

Dr. Maddison, in reference to the taking of cord blood for the Coombs test, said that the easiest way of obtaining blood was by syringe with a fine polythene tube attached; 2 ml. was enough, and no anticoagulant was needed for the test.

The question was asked, "To what extent does post-maturity create neonatal problems?" Dr. Robinson said that he could not answer with certainty. Post-maturity might lead to placental degeneration, but each case had to be decided on its merits, and in general the risk of interference was greater than the risk of post-maturity.

Dr. Maddison, in replying to a question on physiological jaundice, said that the term would probably be discarded as the underlying reasons for post-natal jaundice became more accurately determined. Physiological jaundice as it was known at present was probably associated with deficiencies in liver enzyme systems.

Dr. Robinson, discussing the pathogenesis of hyaline membrane formation, said that it occurred in cases of prematurity, in Cæsarean section and with diabetic mothers, and inhalation of liquor amnii plus exudate from the lining cells of the lung was probably the cause. In reply to questions regarding the treatment of cyanosis, Dr. Robinson said that it was essential in the first place to clear the nasopharynx before the administration of stimulants was considered, and if the airway was clear, then everything possible should be done to improve respiratory effort.

Correspondence.

GENERAL PHARMACEUTICAL BENEFITS.

SIR: I am entirely in accord with the letter of Dr. E. S. Stuckey (MED. J. AUST., January 16, 1960).

There appear to me to be at least three main grounds for complaint:

1. As he pointed out, the virtual dictation of what we may or may not prescribe (if not directly by the Government, at least by the pressure of the patient's tendency in some cases to patronise doctors who will give them "medicine for nothing").

2. The fact that some prescriptions will in fact cost less than the 5s. charged to the patient; e.g., the "National Health Notes for Medical Practitioners" book informs us that one hundred ephedrine hydrochloride one-quarter grain tablets cost 3s.

3. There will be a certain class of patient, who, rather than pay say 10s. 6d. for a bottle of cough mixture, will come to the doctor and say "Give me a prescription for a cough mixture please Doctor" which, in this State with the maximum hospital benefits, costs 10s. 6d. per consultation to the patient (which he may never pay anyway), and he then receives his bottle of cough mixture for 5s.

I would heartily endorse any action taken by the British Medical Association or other body against the proposed scheme.

Yours, etc.,

JOHN B. HUGHES.

1029 South Road,
Burbank,
South Australia.
January 18, 1960.

SIR: May I write in support of Dr. Edward S. Stuckey's letter (Journal, January 16, 1960) referring to the apparent apathy of the profession towards the Commonwealth Government's proposals to greatly extend the scope of general pharmaceutical benefits to the community.

I personally feel very strongly against the proposed "new order", and I venture to say, unbelievable as it may sound, that the Government's scheme is being introduced with the ignorance of 80% of the medical profession. When the above matter was discussed at my local association meeting, 28 of 140 members were present, there being 250 doctors eligible for membership. Is it any wonder that this response is interpreted as apathy?

Is it too late for each individual member of the profession to be circularized with a brief questionnaire requesting his opinion regarding the proposed "new order"?

At the inaugural meeting of the Australian College of General Practitioners held on March 20, 1959, Dr. Ian Grant, President of the College of General Practitioners (British) delivered an address in which he stated that what he had seen of the Australian profession convinced him that we would not be browbeaten into submission by any Government—thereby preventing a recurrence of the disastrous effects of nationalization which have occurred in Britain.

May I conclude by suggesting that it is "now or never"?

Yours, etc.,

MAXWELL G. CHAPMAN.

457 Punchbowl Rd.,
Belfield, N.S.W.
January 18, 1960.

CANCER, A DISEASE OF THE NERVOUS SYSTEM.

SIR: In reply to Dr. Armati's question in THE MEDICAL JOURNAL OF AUSTRALIA, December 19, 1959, I find it necessary to preface my reply with a restatement of the question, as it is an extraordinary question, by no means explicit, in certain respects confused: ". . . whether he considers that the sweating, tachycardia, emotional disturbance, delirium and feelings of collapse which not infrequently precede an attack of pregnancy are convincing evidence that pregnancy is yet another manifestation of autonomic dyspraxia". I presume that the symptoms described occurred during or immediately after the initiating coitus, and that by the term "an attack of pregnancy" Dr. Armati means abnormal pregnancy—pathological pregnancy. I do not consider that the accompaniments and manifestations of an abnormal pregnancy are relevant to a confusion of terms, for autonomic dyspraxia by definition is a pathological condition, and pregnancy is a normal, physiological process. Pathological pregnancies are often a manifestation of autonomic dyspraxia; normal pregnancy never.

Autonomic dyspraxia is a condition of instability of the autonomic nervous system of central origin, caused by an emotional overload, usually fear, and results in the peripheral malfunction of plain-muscle fibre, gland or cell. The supra-threshold emotional stress overwhelms the subcortical autonomic nuclei (S.C.A.N.). The resulting commotio hypothalami prevents these nuclei from carrying out their normal function of coordinating the opposing

sympathetic-parasympathetic activity in various peripheral organs. The resulting abnormal function is pathological, as opposed to normal function being physiological. A normal pregnancy is associated with only normal symptoms and signs.

Unfortunately many pregnancies are pathological. According to the hypothesis a number of these pathological pregnancies are the result of autonomic dyspraxia. Many of the pathological conditions of pregnancy are obviously a failure of autonomic function in a peripheral structure—some cases of spontaneous abortion, some cases of ectopic pregnancy, hyperemesis, perversion of appetite, hypertension, renal failure of certain types and some complication of parturition, e.g., delayed dilatation of the cervix.

Pregnancy is particularly associated with emotional stress—at the beginning, as appears to be the type of case which Dr. Armati had in mind, through to the ordeal of childbirth and its life-long obligations. Whether the emotional stress is supra-threshold or not in any particular case depends on the summation of two variable factors—the genetic weakness of the individual plus the magnitude of the emotional stress. In a case experiencing delirium and a feeling of collapse in the initial stages, this summation of genetic weakness and emotional stress was supra-threshold, caused commotio hypothalami, and peripheral malfunction occurred. In such a case further occurrences of autonomic dyspraxia could be expected throughout the pregnancy. This association between emotional stress and some complications of pregnancy and parturition has been recognized by obstetricians for centuries. I recall very vividly Professor Windeyer reiterating 25 years ago in our obstetric lectures: "A tense woman, a tense cervix." At that time the physiology of the autonomic nervous system, especially its control by the subcortical autonomic nuclei and its inseperable association with emotional stress, were not widely known.

In this type of case, which is relatively simple and where the relevant physiology is well known, the mechanism of autonomic dyspraxia is easily followed. Dilatation of the os in the first stage of labour is a unique process of coordinated plain-muscle activity. The opposing actions of contraction and dilatation are controlled over a period of hours. The whole process is controlled by the autonomic nervous system. The coordination of the opposing peripheral autonomic activities is regulated by the hypothalamus.

Emotional tension acts on the same structure. When the tension exceeds the threshold (fright, anger, or grief), the control of the autonomic nervous system fails (collapse, loss of temper or crying). The same result occurs if the emotional tension is less intense but prolonged, as in frustration and exhaustion. Characteristic electroencephalographic activity is found. So the hypothesis of autonomic dyspraxia is found to explain the physiopathology of the "tense woman, tense cervix" syndrome, at the peripheral level, the central level and the emotional level.

This type of case concerned with plain-muscle malfunction is the easiest to follow. The next step includes cases of glandular malfunction, such as leucorrhœa, then mixed cases (muscle malfunction and gland malfunction) such as peptic ulcer (hypermotility and hyperacidity), and finally the complicated cases at the cellular level (enzymatic malfunction), such as neurosis, allergy, psychosis and possibly neoplasia. So the practice of medicine becomes progressively clearer; out of apparent chaos comes order.

Yours, etc.,

185 Macquarie Street, Sydney.
BRIAN HAYNES.

January 7, 1960.

BLUEPRINT FOR A MODEL PSYCHIATRIC HOSPITAL.

SIR: It was with very real pleasure that I read the historical survey, and exposition of the "present" position (1999), of the mental health services set out by Dr. D. C. Maddison in your Journal (January 9, 1960). I was sorry, however, that he tended to ignore the close association that has grown up over the last 30 or 40 years in this State of the mental health and penal services, the two services recognizing that they are really dealing with exactly the same problem which presents in different ways. For instance, in forensic work, particularly with juvenile delinquents, it is often and rightly said that each case is a "pointer to family abnormality". Further, I was concerned with his reference to an old mental hospital having become a minimum security prison. What happens today (1999) to the mental patient who constitutes a grave risk to the community—a man like Straffen, who lived in the 1950's and 1960's? Surely such persons, who were known as "criminal lunatics", still require considerable security, particularly as 'Drug Trial No. 2651' demonstrated that the various pharmaceuticals are only suitable as short-term ameliorative therapy.

It might interest readers to know that the history outlined by Dr. Maddison as regards the mental hospital has been closely paralleled by that of the penal establishments. We who work in the various prisons and prison hospitals are today also considered to be engaged in a "prestige task", and one of our basic "tools" is the concept of the therapeutic community (a modification of the original model initiated by Maxwell Jones and others in the 1940's and 1950s). Those working in the penal field at that time recognized that the rehabilitation of prisoners was not helped by "the gloom (and) isolation" of the prisons of earlier years and the "conservatism" of many of those that administered them. Equally it was appreciated that there was a necessity for "public education . . . at varying levels of sophistication and complexity", and that cooperation with both general and mental hospitals, with special regard to their specialist facilities, was essential.

Dr. Maddison stresses the importance today of community cooperation, and this is equally recognized in penal circles; as Paterson said so many years ago (1932): "You cannot train men for freedom in conditions of captivity." From quite early in this, now nearly completed, century the concept of probation was officially practised, and later parole in various forms was adopted. Today, those living who remember the 1950's and 1960's must be amazed by the very few wrongdoers sent to prison, even for crimes that were then considered as deserving of the supreme penalty (almost invariably commuted). Now we recognize the futility and expense of sending persons to prison for sentences imposed on an utterly arbitrary basis. Such sentences are not only expensive in themselves (upkeep of establishments, custodial manpower, feeding and clothing of the prisoner, etc.), but also expensive in that the family left at home often becomes a public liability whilst the theoretical breadwinner is only partially, if at all, productively employed. Instead of the penal régime of 40 years ago, we now use fines more effectively (a percentage of the income after reasonable liabilities have been deducted) by employing "attachment" and "distress", as well as having developed the concept of the attendance centre (*Criminal Justice Act*, 1948 s. 48 (2)), where individuals attend during their leisure and learn trades, work in Government workshops, develop hobbies, receive instruction in such things as parenthood and carry out useful civic functions. The concept of deterrence today is recognized as not being achieved through a gross deprivation of liberty, but through social attitudes and a carefully assessed reduction of leisure liberty. The few remaining prisons and prison hospitals house those persons who are considered security risks not at present amenable to treatment (sociopaths), and a few individuals in need of an intensive therapeutic régime under conditions of considerable security. Deterrence still exists as a concept, but we are no longer concerned with the difficult problem of "less eligibility' or "non-superiority" (Mannheim[1]), and the debate regarding the difference, if any, between punishment and treatment has died a natural death.[2]

Again, the courts now might well surprise those who lived some 40 years ago, in that they no longer are the sentencing tribunal, as was the case, and they no longer spend hours fruitlessly interrogating psychiatrists regarding "responsibility" and debating the meaning of rules (M'Naughten) first enunciated in 1843.[3] Today the court endeavours to say whether, within the rules of evidence, the act alleged was committed by the accused; and that is all.[4] If the charge or indictment be proved, the convicted man appears before a tribunal that decides upon the proper mode of disposal after, if necessary, an adequate period of observation and assessment. Anyone who is clearly psychotic or defective is not expected to answer any charge, but is considered *ipso facto* non-culpable, and is cared for and treated in an appropriate institution. This biological definition of insanity was introduced into the Swedish Penal Code in 1945.[5]

A date to be remembered when looking back in the history of penology and social welfare in our State is 1960.

[1] "The Dilemma of Penal Reform", 1939.
[2] *Acta psychiat. (Kbh.)*, 1946, 21: 429.
[3] *Brit. med. J.*, 1954, 2: 713.
[4] *Brit. med. J.*, 1954, 2: 988.
[5] *Brit. J. Delinq.*, 1955, 5: 262.

This was the year when there was enacted provision for a markedly increased unification of the services that pertain to the problem of delinquency and criminality. They were grouped together under the general direction of a central director. This allowed of easy communication between the various departments, and the initiation of more effective longitudinal studies and interdisciplinary research.

Today the penal department and the psychiatric services live most harmoniously together (being nearly one and the same thing), and nearly all the advances Dr. Maddison has noted with respect to the mental hospitals are true for the prisons and prison hospitals. Similarly, all the various "groups" that he has enumerated as being practised in his province are equally practised within the penal framework.

Yours, etc.,

A. A. BARTHOLOMEW.

4 Adamson Street,
Heidelberg,
Victoria.
January 14, 1960.

ACUTE BILATERAL FOOT-DROP IN CHILDREN.

SIR: The report by J. W. Lance (MED. J. AUST., December 26, 1959) is of extreme importance. I became interested in interstitial neuritis 20 years ago in general practice, and later in the Army.[1,2] I discovered that the disorder was more common than I had suspected. Sensory or motor loss may appear after injuries,[3] injections,[4] or infections,[3] or without apparent cause. Complete recovery is common, but a residual defect is usual.

The late Robert Wartenberg collected information on this disorder for more than fifty years.[5] Sensory mononeuritis, he said, is so common that nearly everybody has it once or twice. Two or more sensory nerves may be involved at once or in succession; exact symmetry is common. Motor loss is less prominent but it is common enough—usually combined with sensory but sometimes appearling alone. The patients reported by Dr. Lance show bilateral symmetry of motor lesions. In Case I the injury followed by pain in the shoulder seems significant.

Wartenberg deplores the recent fashion which has thrown the term neuritis into the discard in favour of "neuropathy". Nothing, he says, is gained by any attempts to differentiate inflammatory from degenerative affections of nerve trunks. "From the clinical point of view, neuritis may be defined as a degenerative or inflammatory process in any part of the peripheral nervous system resulting in loss of function."

Peripheral neuritis is usually a trivial disorder; many of the classical descriptions reached the literature because the patients were the doctors themselves or members of the doctor's families. It is not given importance in textbooks, and so any doctor who sees a case may be tempted to believe that he is discovering something new. Most cases of meralgia paræsthetica, tardy ulnar palsy, carpal canal and disk syndromes are in reality the same disorder, which Wartenberg believes to be due to viruses.

Yours, etc.,

MICHAEL KELLY.

410 Albert Street,
East Melbourne.
January 14, 1960.

PREMEDICATION OF CHILDREN.

SIR: Since my letter to you (MED. J. AUST., November 28, 1959), I decided that it was imperative to investigate Dr. I. R. McDonald's statement (MED. J. AUST., October 10, 1959) that, having passed Ryle's tubes on four successive cases premedicated with methylpentynol, the minimum volume aspirated was 8 oz. (230 ml.).

Dr. I. R. McDonald does not state at what interval after the administration of the methylpentynol he passed the Ryle's tube. My technique was that, after induction, and when the child was on the operating table and the Boyle-Davis gag inserted, a small "polythene" œsophageal tube was passed under direct vision. Its presence in the stomach was confirmed by auscultation over the epigastrium. If there was no immediate fluid on aspiration, a few millilitres of water were injected and aspiration repeated. My results were as shown in Table I.

TABLE I.

Age. (Years.)	Premedication.	Time Given.	Time of Aspiration.	Volume.
10	"Elixir Oblivon", 4 dr., hyoscine, 1/100 grain.	8.45	10.15	1 ml.
3	"Elixir Oblivon", 2 dr., hyoscine, 1/150 grain.	8.45	10.15	Nil
7	"Elixir Oblivon", 3 dr., hyoscine, 1/100 grain.	8.45	10.45	Nil
7	"Elixir Oblivon", 3 dr., hyoscine, 1/100 grain.	9.0	11.10	Nil
6	"Elixir Oblivon", 3 dr., hyoscine, 1/100 grain.	9.15	11.30	Nil
12	"Elixir Oblivon", 4 dr., hyoscine, 1/100 grain.	8.30	10.0	10 ml.
6	"Elixir Oblivon", 3 dr., hyoscine, 1/150 grain.	8.45	10.15	Nil
5	"Elixir Oblivon", 2 dr., hyoscine, 1/150 grain.	9.0	10.40	1 ml.

I think these results must once and for all disprove the statement that methylpentynol by mouth causes an increase of gastric secretion with danger of vomiting during induction. But I must again draw attention to the importance of giving the methylpentynol at least one hour and preferably two hours before the induction. Neglect of this fact is, I am sure, the cause of the vomiting when this does occur. And again, may I repeat that the hyoscine is dissolved in the methylpentynol, so that no pre-operative injection is necessary.

Yours, etc.,

F. R. GUSTERSON,
Senior Consultant Anæsthetist.

Worthing Group Hospitals,
Worthing, Sussex,
England.
January, 1960.

PURE PULMONARY VALVE STENOSIS.

SIR: Mr. Harry Windsor's provocative discussion (MED. J. AUST., December 26, 1959) of Dr. M. L. Powell's admirable paper on "Pure Pulmonary Valve Stenosis' (MED. J. AUST., November 28, 1959) invites further comment. Our experience in the Thoracic Surgery Unit of the Alfred Hospital leads us to disagree with his conclusion and feel that there is a very real place for rapid pulmonary valvotomy through the pulmonary artery under hypothermia, or, as we prefer (Orton and Morris, 1959),[1] at normal temperature.

In 16 cases of pulmonary valve stenosis, operated on by trans-arterial pulmonary valvotomy using the fluothane-heparin technique, there have been no deaths. In 12 of these the peak systolic gradient between the right ventricle and pulmonary artery has been reduced to less than 30 mm. of mercury. In four cases, all of them severe, the peak systolic gradient immediately after valvotomy was less satisfactory (Table I). In none of these cases was organic infundibular stenosis observed or felt at the time of valvotomy. There is good reason to believe that regression of the hypertrophy of the outflow tract will occur, and may be expected to result in a further reduction in gradient (Engle *et alii*, 1958;[2] Johnson, 1959[3]). There is clinical evidence of improvement in these cases, supported by changes in the electrocardiogram, phonocardiogram and X ray; but it is our intention to submit these patients to cardiac catheterization one year after valvotomy to confirm or deny this impression.

[1] *Brain*, 1944, 67: 44 (March).
[2] MED. J. AUST., 1946, 2: 325 (September 7).
[3] *Neurology*, 1953, 3: 327 (May).
[4] MED. J. AUST., 1946, 1: 800 (June 8).
[5] "Neuritis. Sensory Neuritis. Neuralagia" (1958), Oxford Univ. Press, New York.

[1] *Thorax*, 1959, 14: 39.
[2] *Circulation*, 1958, 17: 862.
[3] *Brit. Heart. J.*, 1959, 21: 429.

Mr. Harry Windsor further states: "The right ventricle must be opened, the valvotomy done, and sufficient hypertrophied muscle resected to produce an adequate outflow tract with no significant gradient." If the right ventricle is opened and some of its muscle resected, one inescapable consequence is that its function in the post-operative period will be significantly impaired (Stirling, Stanley and Lillehei, 1957[4]). Unless a very complete correction of the stenosis is achieved, this may be fatal. This has been our unhappy experience in one case of extreme pulmonary valve stenosis with marked right ventricular hypertrophy and functional infundibular stenosis, whom we submitted to open operation using total body perfusion. Adequate pulmonary valvotomy resulted in no significant reduction in right ventricular pressure (160 mm. of mercury). The ventricle was opened, and the situation described by Mr. Windsor was observed; the outflow tract was massively hypertrophied and contracted during systole, producing virtual obliteration of the infundibulum. A very radical resection of muscle tissue was carried out, but no significant reduction in gradient was achieved. The patient died 24 hours after operation from right heart failure and unrelieved stenosis.

TABLE I.

Four of 16 Cases, in which the Peak Systolic Gradient was Not Reduced Below 30 mm. of Mercury.

Case.	Patient's Age. (Years.)	Peak Systolic Gradient (Mm. of Mercury).		
		Preoperative Catheterization.	Pressures at Operation.	
			Before Valvotomy.	After Valvotomy.
I	17	92	88	88
II	3	143	120	41
III	4	210	237	81
IV	2	147	150	100

While our experience with resection of the infundibulum in cases of organic infundibular stenosis has been gratifying, it would seem from our limited experience with pulmonary valve stenosis, complicated by severe functional infundibular stenosis, that the decision lies between one of non-interference with the ventricle, on the one hand, and with radical resection combined with prosthetic reconstruction on the other. Until more evidence is available, the first policy seems more appealing.

In summary, while we agree with Mr. Windsor's objective "to produce an adequate outflow tract with no significant gradient", we do not feel sure that this can always be achieved and, if it is not, then the addition of an unnecessary ventriculotomy may prejudice an otherwise favourable outcome.

Yours, etc.,

G. R. STIRLING,
Assistant Thoracic Surgeon.

Thoracic Surgery Unit,
Alfred Hospital,
Melbourne.
December 31, 1959.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE RÖNTGEN RAYS.[1]

[From the *Australasian Medical Gazette*, July 20, 1896.]

DR. F. H. QUAIFE of Woollahra (Sydney) has donated £100 to the Sydney Hospital for the purpose of obtaining the most perfect apparatus for the practical production of the Röntgen rays, to be used in the diagnosis of disease and injury in the interests of suffering humanity—Dr. Quaife has been for some years past Honorary Consulting Physician to the Hospital.

The Röntgen rays have been used successfully at the Melbourne Hospital lately. The patient was a man who had accidentally run a needle into the palm of his right hand. The needle was rusted somewhat, and it rapidly set up inflammation half way along the fore-arm. The location of the needle could not be determined by ordinary means, and Professor Lyle, who had experimented with the Röntgen process, was invited to photograph the hand, and, if necessary, the arm, of the patient, and he did so. The result was quite satisfactory. The shadow-photograph revealed the needle lying across the bones in the palm. Mr. G. A. Syme, one of the Honorary Surgeons, in the presence of the staff, made an incision at the point shown on the photograph and the needle was successfully extracted.

[4] *Surg. Forum*, 8 : 433.

[1] From the original in the Mitchell Library, Sydney.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

PROGRAMME FOR APRIL AND MAY, 1960.

THE following programme of post-graduate courses has been arranged for the months of April and May, 1960.

Week-End Course in Thoracic Diseases.

A week-end course in thoracic diseases suitable for general practitioners will be held on April 2 and 3 at The Royal North Shore Hospital of Sydney. The fee for attendance is three guineas.

Post-Graduate Training in Cardio-Vascular Diseases.

The second of the series of evening seminars on various aspects of cardio-vascular diseases suitable for both general practitioners and post-graduates studying for higher degrees or diplomas will deal with ischæmic heart disease and myocardial infarction. These seminars will be under the supervision of Dr. G. E. Bauer, and will be held in the Maitland Lecture Hall, Sydney Hospital, from 8 to 10 p.m. as follows:

Wednesday, April 6, Introduction: 8 p.m., "Pathology of Ischæmic Heart Disease", by E. S. Finckh; 8.30 p.m., "Fat Metabolism and Ischæmic Heart Disease', Dr. G. V. Hall; 9 p.m., "Prevention of Ischæmic Heart Disease", Dr. H. M. Whyte; 9.30 p.m., recorded lecture, "Functional Pathology of Occlusive Coronary Disease", Dr. Jesse E. Edwards (Mayo Clinic).

Wednesday, April 13, "Coronary Insufficiency": 8 p.m., "Cardiac Pain—Diagnosis', Dr. B. C. Sinclair Smith; 8.30 p.m., "Cardiac Pain—Management", Dr. F. L. Ritchie; 9 p.m., "Treatment of Refractory Angina", Dr. D. S. Stuckey.

Wednesday, April 20, "Myocardial Infarction": 8 p.m., "Typical and Atypical Presentation", Dr. John Raftos; 8.30 p.m., "Diagnostic Aids", Dr. J. G. Richards; 9 p.m., "Complications of Myocardial Infarction", Dr. G. E. Bauer.

Wednesday, April 27, "Treatment": 8 p.m., "Treatment of Myocardial Infarction", Dr. R. G. Epps; 8.30 p.m., symposium on anticoagulant therapy: "Anticoagulants and Laboratory Control", Dr. E. J. Halliday; "Long-Term Anticoagulants", Dr. G. E. Bauer; "Complications and Their Treatment", Dr. W. A. Seldon.

Each seminar will conclude with a panel discussion, and supper will be served. The inclusive fee for attendance is two guineas.

Course for Part I D.D.M.

A course for the Part I examination of the diploma in dermatological medicine of the University of Sydney will begin on May 9 and continue for a period of 12 weeks. Lectures and demonstrations are held in the afternoons only, and the fee for attendance is 50 guineas. Information concerning requirements for this diploma may be obtained from the office of the Committee.

Course for Primary F.R.A.C.S. and F.F.A.R.A.C.S. Examinations.

A part-time refresher course suitable for candidates proceeding to the primary F.R.A.C.S. and F.F.A.R.A.C.S.

examinations, to be held in Sydney on September 1, 1960, has been organized in the Departments of Anatomy, Physiology and Pathology in the University of Sydney, in conjunction with the New South Wales State Committee of the Royal Australasian College of Surgeons. The course will begin on May 9 and continue for a period of 12 weeks, and will take the form of tutorials held in the late afternoons, the last month being devoted to revision and viva-voce examinations. The closing date for applications is March 11, and candidates will assemble shortly after this date to be addressed by selected lecturers and given guidance concerning standard textbooks and reading material in general. Special arrangements will be made for overseas and interstate candidates, who need not attend this preliminary assembly. Applications should be sent to the Post-Graduate Committee. The fee for attendance is 30 guineas.

General Revision Course.

The annual general revision course will be held for two weeks, from Monday to Friday, May 9 to 20, 1960, inclusive. The main theme of the course is therapeutics. As in former years, the programme will be a comprehensive survey of modern trends in diagnosis and treatment of special value to general practitioners, consisting of lectures, special demonstrations, symposia and discussions. Social activities will include a cocktail party, wine tasting and the Thirteenth Annual Post-Graduate Oration in the Great Hall of the University of Sydney, to be given by Dr. George Willcocks on "The Sydney Hospital Group at the Turn of the Century", to which all members of the course and their wives are invited. A golf competition for the Post-Graduate Golf Cup open to members will also be held, and the Brydon Cup will be for competition by country members.

The fee for attendance is twelve guineas full-time or six guineas for one week or part-time. Fees and travelling expenses incurred by practising members of the medical profession in attending this course may be claimed as income tax deductions.

Financial Assistance to Practising Doctors in Isolated or Remote Areas of Australia.

The Post-Graduate Committee in Medicine has set aside a sum made available by the Post-Graduate Medical Foundation, to assist practising doctors in isolated or remote areas in Australia towards the payment of fares and fees in attending the general revision course in 1960. Grants up to £50 per person will be made. Early application is essential.

Method of Enrolment.

Early written application to attend the above-mentioned courses should be made to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney".

SEMINARS AT THE ROYAL NORTH SHORE HOSPITAL OF SYDNEY.

THE following is the programme of seminars at the Royal North Shore Hospital of Sydney from February to June, 1960.

February 2: "Recent Advances in Pulmonary Function Tests", Dr. J. R. Read.

March 1: "Recent Advances in Clinical Biochemistry", Dr. F. C. Neale and Dr. F. J. Radcliff.

April 5: "Recent Advances in Virus Diseases", Professor P. de Burgh.

May 3: "Recent Advances in the Virilizing Syndromes", Associate Professor R. P. Shearman.

June 7: "Recent Advances in Gastroenterology", Dr. A. Skyring.

The seminars will be held on the first Tuesday of each month at 5.15 p.m. in the Students' Common Room. On the remaining Tuesdays at this time, a clinical demonstration is held in B2 Tutorial Room. It has been decided this year to conduct initially a "Recent Advances" series.

SEMINARS AT SYDNEY HOSPITAL.

THE following seminars will be held at Sydney Hospital during March and April, 1960.

March 2: "Acute Leukæmia", Dr. T. I. Robertson, Hæmatology Clinic.

March 9: "Assessment and Treatment of Valvular Stenosis in the Heart", Professor R. B. Blacket, University of New South Wales.

March 16: "Iron and the Liver", Dr. B. R. M. Hurt, Gastro-Enterology Clinic.

March 23: "Metabolic Studies in Obesity", Dr. B. C. E. Ashley, Clinical Research Unit.

March 30: "Some Aspects of Ulcerative Colitis", Dr. Bruce Hall, St. Vincent's Hospital.

April 6: "Myasthenia Gravis", Dr. Francis F. Foldes, Physician-in-Charge of Myasthenia Gravis Treatment Center, Mercy Hospital, Pittsburgh, Pennsylvania.

April 13: "Problems in the Diagnosis of Rheumatic Fever", Professor L. Dods.

April 20: "Hypoparathyroidism", Dr. L. C. A. Watson, Endocrine Clinic.

April 27: "Sarcoidosis", Dr. J. E. Hassell (by invitation), Thoracic Clinic.

The seminars will be held on Wednesdays from 2 to 3 p.m. in the Maitland Lecture Theatre.

Obituary.

DAVID MURRAY MORTON.

WE are indebted to Dr. Raymond Hennessy for the following account of the career of the late Dr. Murray Morton.

David Murray Morton died on September 30, 1959, after a brief attack of angina pectoris, six days after reaching his eighty-eighth birthday. Until his final illness he had enjoyed excellent health, was in entire possession of his mental faculties and had none of the physical disabilities usual at his age. As he resigned from his hospital in 1932 and retired from practice in 1940, to several generations of practitioners he was known only by hearsay. Nevertheless, all his old pupils lamented the passing of a highly esteemed and accomplished surgeon.

He matriculated at the early age of 14 years, and was unable to begin his medical course until he was 16 years of age. He graduated with honours in 1892. He won the medical exhibition worth £70 and was runner-up for the Beaney Prize, which then was competed for as a separate examination. He served one year as resident medical officer at the Melbourne Hospital, and then for two and a half years he was resident at the Alfred Hospital, at this time not a recognized clinical school. There was no salary from the Melbourne Hospital, but the Alfred Hospital gave £100 *per annum*.

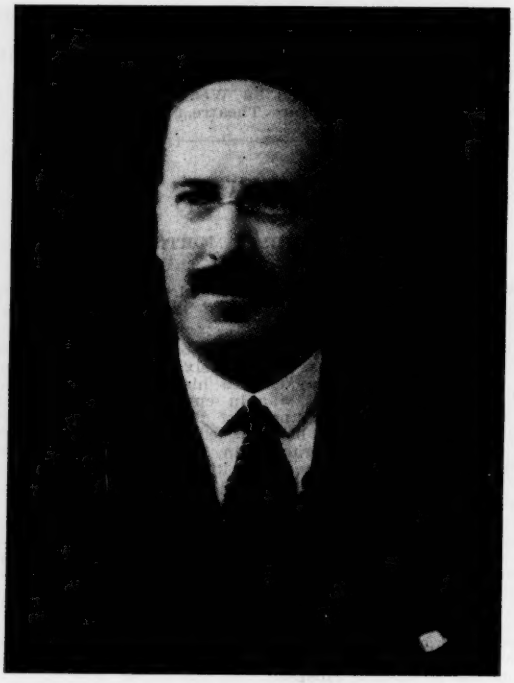
In 1896 he began practice in Richmond, where he remained until 1909. He then relinquished general practice, and after a visit to the various medical centres in Europe, England and the U.S.A., he set up as a consulting surgeon. He had obtained the M.D. degree in 1899.

Dr. Morton joined the staff of St. Vincent's Hospital as an anæsthetist in 1896, three years after the foundation of the hospital. His active association with the hospital continued without interruption until 1932, when he was appointed Consulting Surgeon. St. Vincent's Hospital became a clinical school in 1910, and Dr. Morton was the first Dean of the Clinical School. He performed the first operation in the new wing when it was opened in 1905. He was a foundation Fellow of the Royal Australasian College of Surgeons, and was also a Fellow of the American College of Surgeons. He was for many years an examiner in surgery at the University of Melbourne.

Dr. Morton's career as student and graduate covered a long period dating from the Listerian era. When he was a student he actually saw the carbolic spray in use in the operating theatre. When he first joined St. Vincent's Hospital, the operating room was the main passageway through the building in the upper floor. Nevertheless, it is on record that in his first year at the hospital 202 operations were performed. He may have been the last survivor who saw Beaney operate; the operation was for an abdominal aneurysm. He also attended the autopsy on the patient the following day. At the time of his graduation, the frock coat and high hat were the essential apparel of the doctor. The wooden stethescope had not

yet been supplemented by the rubber binaural instrument, and was conveniently carried in the high hat.

Dr. Morton was one of the most fastidiously clean people I have ever known, and I think that the methods of aseptic surgery would come naturally to him; his technique in this respect was literally perfect and consistently correct. As an operator he was neat, tidy and thorough, and at a set operation, like a radical mastectomy or herniotomy, he had no superior. He was an excellent teacher, eminently practical; his delivery was good, and he expressed himself in clear, precise language spiced with a pleasant wit. He was a man of incorruptible probity and scrupulous fairness, and was in much demand as a counsellor to any of his colleagues in trouble. These qualities, universally acknowledged, made him a most valuable member of the Medical Defence Association, of which he was president for many years.



He was one of the first Australian surgeons to practise Arbuthnot Lane's open operative treatment of simple fractures. He was very successful with the method, which made a great appeal to him on account of the exaggerated aseptic technique it required.

He was below average height, with an elegant figure. He dressed with impeccable taste, and his style and deportment were those of a well-bred gentleman, which indeed he was. He was a delightful postprandial orator and a charming raconteur. Few people could tell a story better.

On his retirement from practice, many of his old pupils and colleagues entertained him at dinner and presented him with a silver salver engraved with their individual autograph. They also commissioned the well-known artist Colin Colahan to paint his portrait, which hangs prominently in the board room of the hospital he served long and faithfully for more than half a century. It is an excellent likeness and an acknowledged work of art.

Dr. Morton had a fund of pleasant anecdotes about his early medical life and colleagues. As a young man, his heroes were Sir Thomas Fitzgerald and Henry O'Hara. He used to recall how his rise to affluence was indicated by his means of transport; first a bicycle, then a gig, then a buggy, which he had shod with rubber tyres, amongst the first to be so in Melbourne. He was able eventually to put his groom into full livery, with metal buttons, breeches, top hat, top boots and driving gloves, and thus he drove around the streets of Richmond in all the splendour of a successful practitioner. Professor Halford, the founder of the Melbourne Medical School in 1864, was one of his teachers. His chief recollection of the professor was his constant habit of repeating "God bless the man who invented beer and bread and cheese"—a sentiment with which he said he was in entire accord.

Dr. Morton was fond of dabbling in medico-legal affairs. This was because he had been an unfortunate but successful defendant in a legal action early in his career, which he always said turned him subsequently into a bush lawyer. Dr. Morton was twice married, and he is survived by a daughter of the first marriage and a widow.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 83, of December 23, 1959.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—4/31906 Major (Temporary Lieutenant-Colonel) M. W. Elliott is appointed to command 11th Field Ambulance, 2nd January, 1960. 1/39189 Lieutenant-Colonel V. E. Sampson, M.C., relinquishes command 11th Field Ambulance, 1st January, 1960. The provisional rank of 1/46919 Captain G. R. C. McLeod is confirmed.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—2/122031 Captain (provisionally) H. W. Thurlow relinquishes the provisional rank of Captain, 15th October, 1959, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command) and is granted the honorary rank of Captain, 16th October, 1959. The age for retirement of 2/147950 Lieutenant-Colonel C. E. M. Gunther is extended until 3rd February, 1961. The age for retirement of 2/206951 Colonel E. F. Thomson is extended until 1st January, 1962.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The provisional appointments of the following officers are terminated:—Captains 3/50197 W. A. Syme, 26th July, 1959, and 3/111532 H. A. Marks, 3rd August, 1959. *To be Captains (provisionally)*—3/50197 William Archibald Syme, 27th July, 1959, and 3/111532 Howard Alfred Marks, 4th August, 1959.

Central Command.

Royal Australian Army Medical Corps (Medical).—4/32082 Captain (provisionally) L. L. Hoare relinquishes the provisional rank of Captain, 10th November, 1959, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Central Command) in the honorary rank of Captain, 11th November, 1959. *To be Major, 16th November, 1959*—4/32055 Captain (Temporary Major) R. Hunter.

Western Command.

Royal Australian Army Medical Corps (Medical).—5/10736 Captain G. M. Nunn relinquishes the temporary rank of Major, 2nd November, 1959, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Western Command), 3rd November, 1959. The provisional appointment of 5/38095 Captain S. S. Gubbay is terminated, 28th November, 1959. *To be Captain (provisionally), 29th November, 1959*—5/38095 Sasson Stephen Gubbay.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Central Command.—Honorary Captain O. W. Frewin is retired 31st December, 1959.

Central Command.—The following officer is placed upon the Retired List (Central Command) with permission to retain his rank and wear the prescribed uniform, 31st December, 1959: Captain J. H. Petchell.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The following Flight Lieutenants (Acting Squadron Leaders) are promoted to the rank of Squadron Leader:—

E. R. Bowler (0310759), W. I. Stuart (0310760), G. C. Nelson (018487).

Air Force Reserve.

Medical Branch.

Brian Francis William Quinn (0212389) is appointed to a commission, 20th October, 1959, with the rank of Flight Lieutenant.

The appointment of the following officers is terminated:—Squadron Leaders N. Y. McCallum (277624), L. A. Watson (034058); Flight Lieutenants W. B. Law (277520), J. McD. Quinlivan (051379), A. W. Raymond (025592), C. R. Wherrett (267599), W. W. Wilson (018293), 1st October, 1959; R. F. Zacharin (257843), 1st November, 1959.

Notes and News.

Television in the Presentation of Dental Clinics.

The Executive of the Sixteenth Australian Dental Congress has arranged a demonstration of the use of television in presenting clinics to a large number of viewers. The demonstration will be held at the Stawell Hall, 145 Macquarie Street, Sydney, on Thursday, February 4, 1960, at 3 p.m. All members of the British Medical Association who are interested are invited to be present.

WHO: Appointment of Dr. A. J. Metcalfe.

The Director-General of the Commonwealth Department of Health, Dr. A. J. Metcalfe, has been appointed Chairman of the Standing Committee on Administration and Finance of the Executive Board of the World Health Organization. The Commonwealth Minister for Health, Dr. the Honourable D. A. Cameron, in announcing the appointment, said that it was a tribute to Dr. Metcalfe's work for WHO and to the Australian Government's consistent interest in the activities of the organization.

The committee of which Dr. Metcalfe has become chairman is a committee of seven members of the Executive Board of WHO. The Executive Board itself comprises 18 members, all representative of WHO member-countries. The committee meets prior to the session of the Executive Board, to make detailed analyses of the financial aspects of the proposed annual budget estimates, as submitted by the Director-General of WHO. The Executive Board discusses these proposals, in the light of the Standing Committee's recommendations, before referring them to the World Health Assembly for final acceptance or rejection. As a result of Dr. Metcalfe's appointment, the Australian Government has been brought into intimate contact with the whole financial structure of WHO.

Eighth Congress of the Pan-Pacific Surgical Association.

The eighth Congress of the Pan-Pacific Surgical Association will be held at Honolulu, Hawaii, from September 27 to October 5, 1960. All members of the medical profession are eligible to register, and because of limited space they are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities. An outstanding scientific programme by leading surgeons promises to be of interest to all doctors. Ten surgical specialty sections are held simultaneously. Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director-General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

International Society of Cybernetic Medicine.

The International Society of Cybernetic Medicine, constituted in 1958 with the participation of 19 countries, held its first general assembly at Naples in November, 1959. The following office bearers were elected: President, Professor Aldo Masturzo, of Naples University; Vice-President, Professor Paul Nayrac, of Lille University; members of the Council: Professor N. Wiener, Professor G. Asboe-Hansen, Professor F. Nember, Professor A. Gata, Professor C. Coruzzi. It has been decided to organize an Inter-

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 26, 1959.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	1	2(1)	..	..	..	..	..	4
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	6	..	6
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3	5(5)	1(1)	..	1	..	3	..	13
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	..	2(2)	..	1(1)	..	..	..	3
Encephalitis	1	..	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	2	..	..	2
Infective Hepatitis	45(18)	14(3)	8(1)	12(6)	9(4)	..	..	1	89
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	1	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	3(1)	..	..	..	..	..	3
Meningococcal Infection	1(1)	..	..	1(1)	..	1(1)	..	..	3
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	1(1)	..	..	..	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2(1)	..	..	1(1)	..	..	..	..	3
Puerperal Fever	..	1	..	..	..	..	..	..	1
Rubella	..	8(3)	..	..	..	..	..	1	9
Salmonella Infection	..	..	..	7(4)	..	..	..	..	7
Scarlet Fever	9(2)	8(6)	2(1)	4(4)	..	2	..	..	25
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	1	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	22(11)	3(2)	15(5)	1(1)	9(4)	3(1)	..	..	53
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

national Symposium on Cybernetic Medicine at Naples in 1960, with the collaboration of Professor N. Wiener. The office of the Secretary-General is situated at Via Roma 348, Naples, Italy.

Prize for a Medico-Surgical Film.

The annual prize awarded by *La Presse médicale* for a medico-surgical film will be presented on March 22 instead of April 5, 1960, as was announced in the Journal on November 21, 1959 (page 781). The closing date for receipt of entries is now February 15, 1960, not February 29, as previously indicated. Details of the competition are given in the previous announcement.

First National Conference of Social Welfare.

The Australian Council of Social Service announces that the first National Conference of Social Welfare will be held in Melbourne from May 23 to 27, 1960. The theme will be "Social Welfare in Australia—Today and Tomorrow", and the comprehensive programme has been planned for the benefit of all interested in social welfare. The conference is open to all member organizations of the Australian Council of Social Service, and to all interested in social welfare. Correspondence should be addressed to the Organizing Secretary, Mrs. G. Myles, B.A., 44 Beaver Street, East Malvern, S.E. 5, Melbourne. Telephone: *211-3036.*

Medical Appointments.

THE undermentioned appointments have been made at the Royal Adelaide Hospital, Adelaide.

Dr. R. B. Jones has been appointed Honorary Clinical Assistant in the Department of Surgery.

Dr. D. M. Branson has been appointed Honorary Clinical Assistant in the Ophthalmic Department.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Francis, John Fulton, M.B., B.S., 1958 (Univ. Sydney), *Royal Newcastle Hospital, Newcastle.*

Seaton, Douglass George, M.B., B.S., 1954 (Univ. Sydney), F.R.C.S., Edinburgh, 1959, 486 Old South Head Road, Rose Bay.

Chariw, Peter, M.D., 1947 (Univ. Vienna) (licensed *under the provisions of Section 21c of the Medical Practitioners Act*, 1938-1958), 27 James Street, Leichhardt.

Fixler, Sylvia, *M.B., B.S., 1959 (Univ. Sydney)*, 7 Kenneth Street, Bondi.

The undermentioned have applied for election as members of the Victorian Branch of the British Medical Association:

Halley, Winifred, M.B., B.S. (Adelaide), Nyahwest, *Victoria.*

Langley, Jennifer, M.B., B.S. (Sydney), McCallum Road, Doncaster, Victoria.

Deaths.

THE following deaths have been announced:

CEREXHE.—Francis Gordon Archer Cerexhe, on January 13, 1960, at Randwick, New South Wales.

FRANKLIN.—Samuel de Vere Franklin, on January 7, 1960, at Mandurah.

Diary for the Month.

FEBRUARY 2.—New South Wales Branch, B.M.A.: Organisation and Science Committee.
FEBRUARY 3.—Western Australian Branch, B.M.A.: Branch Council.
FEBRUARY 4.—South Australian Branch, B.M.A.: Council Meeting.
FEBRUARY 5.—Queensland Branch, B.M.A.: General Meeting.
FEBRUARY 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEBRUARY 11.—New South Wales Branch, B.M.A.: Public Relations Committee.
FEBRUARY 12.—Queensland Branch, B.M.A.: Council Meeting.
FEBRUARY 12.—Tasmanian Branch, B.M.A.: Branch Council.
FEBRUARY 15.—Victorian Branch, B.M.A.: Finance Sub-committee.
FEBRUARY 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

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ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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